Über dieses Buch

Dies ist ein digitales Exemplar eines Buches, das seit Generationen in den Regalen der Bibliotheken aufbewahrt wurde, bevor es von Google im Rahmen eines Projekts, mit dem die Bücher dieser Welt online verfügbar gemacht werden sollen, sorgfältig gescannt wurde.

Das Buch hat das Urheberrecht überdauert und kann nun öffentlich zugänglich gemacht werden. Ein öffentlich zugängliches Buch ist ein Buch, das niemals Urheberrechten unterlag oder bei dem die Schutzfrist des Urheberrechts abgelaufen ist. Ob ein Buch öffentlich zugänglich ist, kann von Land zu Land unterschiedlich sein. Öffentlich zugängliche Bücher sind unser Tor zur Vergangenheit und stellen ein geschichtliches, kulturelles und wissenschaftliches Vermögen dar, das häufig nur schwierig zu entdecken ist.

Gebrauchsspuren, Anmerkungen und andere Randbemerkungen, die im Originalband enthalten sind, finden sich auch in dieser Datei – eine Erinnerung an die lange Reise, die das Buch vom Verleger zu einer Bibliothek und weiter zu Ihnen hinter sich gebracht hat.

Nutzungsrichtlinien

Google ist stolz, mit Bibliotheken in partnerschaftlicher Zusammenarbeit öffentlich zugängliches Material zu digitalisieren und einer breiten Masse zugänglich zu machen. Öffentlich zugängliche Bücher gehören der Öffentlichkeit, und wir sind nur ihre Hüter. Nichtsdestotrotz ist diese Arbeit kostspielig. Um diese Ressource weiterhin zur Verfügung stellen zu können, haben wir Schritte unternommen, um den Missbrauch durch kommerzielle Parteien zu verhindern. Dazu gehören technische Einschränkungen für automatisierte Abfragen.

Wir bitten Sie um Einhaltung folgender Richtlinien:

+ *Nutzung der Dateien zu nichtkommerziellen Zwecken* Wir haben Google Buchsuche für Endanwender konzipiert und möchten, dass Sie diese Dateien nur für persönliche, nichtkommerzielle Zwecke verwenden.

+ *Keine automatisierten Abfragen* Senden Sie keine automatisierten Abfragen irgendwelcher Art an das Google-System. Wenn Sie Recherchen über maschinelle Übersetzung, optische Zeichenerkennung oder andere Bereiche durchführen, in denen der Zugang zu Text in großen Mengen nützlich ist, wenden Sie sich bitte an uns. Wir fördern die Nutzung des öffentlich zugänglichen Materials für diese Zwecke und können Ihnen unter Umständen helfen.

+ *Beibehaltung von Google-Markenelementen* Das "Wasserzeichen" von Google, das Sie in jeder Datei finden, ist wichtig zur Information über dieses Projekt und hilft den Anwendern weiteres Material über Google Buchsuche zu finden. Bitte entfernen Sie das Wasserzeichen nicht.

+ *Bewegen Sie sich innerhalb der Legalität* Unabhängig von Ihrem Verwendungszweck müssen Sie sich Ihrer Verantwortung bewusst sein, sicherzustellen, dass Ihre Nutzung legal ist. Gehen Sie nicht davon aus, dass ein Buch, das nach unserem Dafürhalten für Nutzer in den USA öffentlich zugänglich ist, auch für Nutzer in anderen Ländern öffentlich zugänglich ist. Ob ein Buch noch dem Urheberrecht unterliegt, ist von Land zu Land verschieden. Wir können keine Beratung leisten, ob eine bestimmte Nutzung eines bestimmten Buches gesetzlich zulässig ist. Gehen Sie nicht davon aus, dass das Erscheinen eines Buchs in Google Buchsuche bedeutet, dass es in jeder Form und überall auf der Welt verwendet werden kann. Eine Urheberrechtsverletzung kann schwerwiegende Folgen haben.

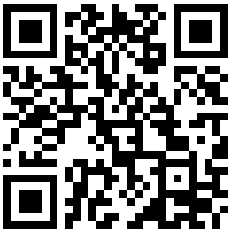
Über Google Buchsuche

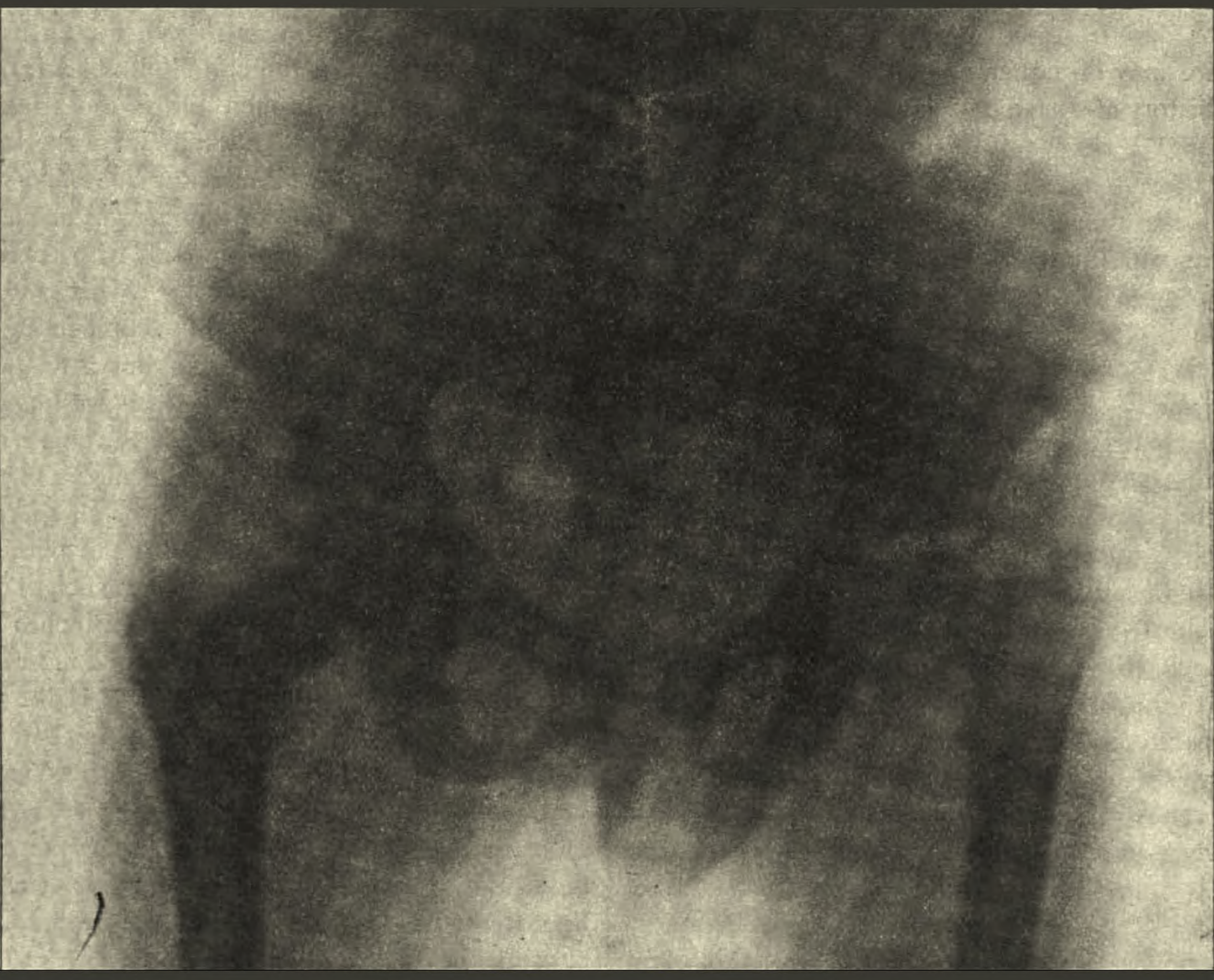
Das Ziel von Google besteht darin, die weltweiten Informationen zu organisieren und allgemein nutzbar und zugänglich zu machen. Google Buchsuche hilft Lesern dabei, die Bücher dieser Welt zu entdecken, und unterstützt Autoren und Verleger dabei, neue Zielgruppen zu erreichen. Den gesamten Buchtext können Sie im Internet unter `http://books.google.com` durchsuchen.

This is a reproduction of a library book that was digitized by Google as part of an ongoing effort to preserve the information in books and make it universally accessible.

Google™ books

https://books.google.com





The Clinical journal

Digitized by Google

UNIVERSITY OF CALIFORNIA
MEDICAL CENTER LIBRARY
SAN FRANCISCO

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

THE

CLINICAL JOURNAL

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE, AND CLINICAL REVIEW.

A WEEKLY RECORD OF CLINICAL MEDICINE AND SURGERY, WITH THEIR SPECIAL BRANCHES.

IN TWO VOLUMES ANNUALLY.

VOL. XXIII.

OCTOBER 21, 1903—APRIL 13, 1904.

TWELFTH YEAR.

EDITED BY

L. ELIOT CREASY, M.R.C.S.ENG., L.R.C.P.LOND.

THE MEDICAL PUBLISHING COMPANY, LIMITED,
22½ BARTHOLOMEW CLOSE, LONDON, E.C.

MCMIV.

Digitized by Google

UNIV OF CALIF
MEDICAL SCHOOL

Digitized by Google

LIST OF CONTRIBUTORS.

PAGE

BARNARD, HAROLD L., M.S., F.R.C.S. A Clinical Demonstration 188
BENNETT, SIR WILLIAM, K.C.V.O., F.R.C.S. A Clinical Lecture on Some Points relating to Rigidity of the Spine 49
BISHOP, E. STANMORE, F.R.C.S. Proliferating Papillomatous Ovarian Cysts 329
BOWLBY, ANTHONY, C.M.G., F.R.C.S. Pain: Its Importance in Diagnosis and its Tendency to Mislead 289
BRADFORD, JOHN ROSE, M.D., F.R.C.P., F.R.S. The Value of Symptoms in Diagnosis ... 193
BRAMWELL, BYROM, M.D., F.R.C.P.E. A Demonstration of Cases at the Polyclinic ... 237, 254
BROOK, W. H. B., M.D., F.R.C.S. On the Treatment of Appendicitis 413
BRYANT, J. H., M.D., F.R.C.P. Two Clinical Lectures on Aneurysm of the Abdominal Aorta 71, 89
BUSHNELL, FRANK, M.D. Large Mononuclear Leucocytosis in Malaria, Blackwater Fever, and Lymphadenoma... 59
BUTLIN, H. T., F.R.C.S., D.C.L. A Clinical Lecture on Difficulty in Swallowing 1
„ „ „ On Predisposing and Pre-Cancerous Conditions of the Breast 337

CAUTLEY, EDMUND, M.D., F.R.C.P. Four Lectures on Normal and Abnormal Conditions of the Blood 343, 362, 380, 395
CHAPMAN, CHARLES W., M.D., M.R.C.P. At the National Hospital for Diseases of the Heart 349
CHEYNE, W. WATSON, C.B., F.R.C.S., F.R.S. Thoroughness in Surgery... 33
CLARKE, J. JACKSON, M.B., F.R.C.S. Two Lectures on Piles, their Nature and Treatment 177, 206
COLLINS, E. TREACHER, F.R.C.S. On Injuries and Diseases of the Orbit 241

DORAN, ALBAN, F.R.C.S. A Clinical Lecture on Broad Ligament Tumours 149
DUCKWORTH, SIR DYCE, M.D., F.R.C.P. A Clinical Lecture on Varieties of Arthritis 113

FENWICK, W. SOLTAU, M.D., M.R.C.P. Hyperæsthesia of the Stomach 296
FOX, T. COLCOTT, M.B., F.R.C.P. A Clinical Lecture on Impetigo 214
FRANCIS, ALEXANDER, M.B., B.C. On the Nature of the Connection between Asthma and the Nose 222

HABERSHON, S. H., M.D., F.R.C.P. Two Lectures on Bronchiectasis 161, 183
HERSCHELL, GEORGE, M.D. On the Differential Diagnosis of Chronic Disorders of the Stomach 385
HUTCHISON, ROBERT, M.D., F.R.C.P. A Clinical Lecture on Some Common Symptoms of Disease in Children and their Diagnostic Significance 353

LACK, H. LAMBERT, F.R.C.S. A Lecture on Nasal Polypus 8
„ „ „ A Lecture on Ozæna or Atrophic Rhinitis 145
LANE, W. ARBUTHNOT, M.S. On the Operative Treatment of the Conditions of the Large Bowel which result from Chronic Constipation 209
LEGG, T. P., F.R.C.S. Three Clinical Lectures on Head Injuries 137, 153, 166

Digitized by Google

PAGE

McGAVIN, LAWRIE, F.R.C.S. The Treatment of Tuberculous Disease of the Tarsus 300

MEACHEN, G. NORMAN, M.D., B.S., M.R.C.P. A Clinical Lecture on Itching as a Symptom, and its Treatment 121

MORTON, CHARLES A., F.R.C.S. The Diagnosis of the Cause of Acute Abdominal Pain ... 232

MOTT, F. W., M.D., F.R.C.P., F.R.S. A Clinical Lecture on Spleno-Medullary Leukæmia ... 225

OGILVIE, GEORGE, B.Sc., M.B., M.R.C.P. What is Syphilitic Disease of the Nervous System? 401

PAGET, STEPHEN, F.R.C.S. On Some Aural Cases 107

PEARSON, S. VERE, M.B., M.R.C.P. Cases Illustrating some Rarer Types of Pneumonia in Infants 200

POWER, D'ARCY, F.R.C.S. In the Out-patient Department of St. Bartholomew's Hospital ... 13

" " A Lecture on Some Cases of Gastric Surgery and their Results ... 97

PYE-SMITH, P. H., M.D., F.R.C.P., F.R.S. A Lecture on Albuminuria 129

ROBSON, A. W. MAYO, F.R.C.S. The Surgical Treatment of Gastric Ulcer 311

SAVAGE, GEORGE, M.D., F.R.C.P. Two Lectures on General Paralysis of the Insane ... 321, 369

SHAW, H. BATTY, M.D. Non-Tuberculous Apical Affections of the Lungs 377

SHEILD, A. MARMADUKE, M.B., F.R.C.S. A Demonstration of Cases at St. George's Hospital 65

" " " " A Clinical Lecture on the Diagnosis and Treatment of Hæmorrhoids 273

SHERREN, JAMES, F.R.C.S. A Clinical Lecture on Some Common Nerve Injuries, their Examination and Diagnosis 53

SMITH, FRED. J., M.D., F.R.C.P. Post-Graduate Lecture on Emphysema and Bronchitis ... 43

" " " A Clinical Lecture on Giddiness 306

STILL, GEORGE F., M.A., M.D., F.R.C.P. A Lecture on the Modification of Cow's Milk for Infant-Feeding 81

TAYLOR, JAMES, M.D., F.R.C.P. A Clinical Lecture on Epilepsy and its Treatment 26

THOMSON, H. CAMPBELL, M.D., F.R.C.P. Clinical Studies in Diseases of the Nervous System: General Paralysis of the Insane 119

" " " The Trophic Changes in Locomotor Ataxy .. 264

" " " Case of Injury to the Cerebral Cortex 392

WHITE, W. HALE, M.D., F.R.C.P. Some Clinical Considerations concerning Certain of the Rarer Forms of Enlargement of the Heart 17

" " " On Common Diseases of the Liver 257

WILLIAMS, LEONARD, M.D., M.R.C.P. A Lecture on Some Points in Balneary Therapeutics 249

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 573. WEDNESDAY, OCTOBER 21, 1903. Vol. XXIII. No. 1.

CONTENTS.

PAGE

*A Clinical Lecture on "Difficulty in Swallowing." By H. T. BUTLIN, F.R.C.S., D.C.L. 1

*A Lecture on Nasal Polypus. By H. LAMBERT LACK, F.R.C.S. 8

*With Mr. D'Arcy Power in the Out-patients' Department of St. Bartholomew's Hospital 13

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½, *Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

ON

"DIFFICULTY IN SWALLOWING."

Delivered at St. Bartholomew's Hospital.

By H. T. BUTLIN, F.R.C.S., D.C.L.,
Consulting Surgeon to the Hospital.

GENTLEMEN,—I have long made up my mind what shall be the subject of my lecture next Wednesday, but I was in great doubt as to what I would lecture on to-day. I wished to give what I am in the habit of calling "a house lecture,"—that is to say, not elaborate enough to publish, but on the other hand often more valuable to students of the hospital who are in the habit of watching its practice. Being in doubt, I went to my house surgeon, and I said, "What shall I lecture upon?" He considered the matter, and next day said, "Why not lecture upon *difficulty in swallowing*, in connection with that patient who is in Paget Ward at the present time?" At first I was not very much attracted by the subject, but the more I considered it the more I thought I might be able to make a useful lecture out of it.

You may remark at the outset that I am lecturing now not on a disease, but on a symptom, a symptom which may be due to a very large number of different causes. It would be impossible in a single lecture to consider every cause of difficulty in swallowing, and in order to limit the subject within reasonable bounds I do not propose to take any of the acute difficulties, or any of the acute troubles which cause difficulty in swallowing. I am going to take chronic dysphagia, the cases in which difficulty in swallowing has existed for a considerable time, and I shall consider that subject alone. Of course it will be impossible for me to lay down definite laws for diagnosis and prognosis in cases of chronic difficulty in swallowing; but at the same time I think I may aid you in making a diagnosis more readily

Digitized by Google

than you otherwise might do. I shall treat the matter almost in the form of aphorisms, and I shall illustrate it by relating cases which have either occurred to myself or in which I have borne a part.

We will take in the first instance *age* in relation to difficulty in swallowing of a chronic character. You can make up your mind that in children and young people—I think you may fairly say up to the age of eighteen or twenty—chronic dysphagia is very rare; and if it is met with it is generally due to some very unusual cause. Of course quite young people may suffer from tubercle of the pharynx and larynx, and that may give rise to difficulty and pain in swallowing, but that is not very common. I can recall at this moment two cases of difficulty in swallowing in young people, both very unusual. First, there was brought to me some years ago, at my house, a boy aged eleven or twelve, and I learnt from his mother that never in his life had he been able to swallow solid food. He was said to be able to take fluids as well as anybody else, but could not swallow solids. Yet he was well, and seemed to be well nourished and developed. I examined his mouth and throat very carefully, and of course I felt his neck and used the laryngoscope. Finding nothing there, I made up my mind to pass an œsophageal bougie. But that can only be accomplished satisfactorily in these young people under an anæsthetic. That very afternoon, at my house, Mr. Gill gave the boy chloroform, and I passed the bougie. About the middle of the œsophagus it seemed to encounter some very slight hindrance; and overcoming that hindrance by a little pressure, I passed it into the stomach. Before the boy had thoroughly recovered from the anæsthetic I put a lump of bread into his mouth, and he swallowed it down without difficulty; but as soon as he came out of the anæsthetic, and had thoroughly regained his senses, I could not persuade him to try to swallow even the smallest piece of bread or any other solid. So he left my house no better able to swallow, in his own mind, than when he came. But I am certain that the obstruction had been broken down, and that he really could have swallowed well. Second, there was a patient in this hospital about fifteen months ago. She was fifteen years of age, and very small; she looked quite an infant. She was emaciated to an extraordinary degree, and her weight was very little. The history which I received was that never in her life had she been able to swallow any kind of solid food, and that she swallowed even fluids with some difficulty; and consequently she had become more and more emaciated and had long ceased to grow, and at length it seemed as if she must die of starvation. I thought it was possible she might have a somewhat similar condition to the boy whose case I have just related to you. So after making an examination of the interior of the mouth and throat and finding nothing there, I had her placed under the influence of an anæsthetic, and passed an instrument down to the middle of the œsophagus, where there was evidently some obstruction; but I passed a bougie of moderate size through this obstruction, and it went down into the stomach. I thought I might have to repeat the operation several times, and that I should never be able to do it without an anæsthetic; and as the child was in a very weak and miserable state I thought it would be best to pass a soft tube down through the obstruction into the stomach, and leave the end projecting from the mouth and feed her through it for a few days, until she became stronger. I also hoped that by keeping the instrument in, although it was quite soft, I could secure a more permanent dilatation of her stricture, if it was a stricture. She was so ill from the presence of the tube, and so constantly sick, that, had it not been for the incessant care and nursing of Sister Paget, she would have died. To my disappointment I was very soon obliged to take the tube out. She was so reduced and in such a miserable state, and I was so hopeless about her, that I had it in my mind to perform gastrostomy, and I should have done so if she had been in better condition. However, she was not able to bear the shock even of a modern gastrostomy. So by great good fortune I did not perform the operation; for from that time she began to mend, she began to take fluids, and afterwards she commenced taking soft substances like jelly and blanc-mange. At the end of two or three weeks she was able to swallow large boluses of soft solid food; until she left the hospital able to take food like any other child. In that case, again, I think I broke down a congenital web, and the child is practically cured. She gained flesh with great rapidity, and grew. Here is a specimen which may help to explain the

Digitized by Google

occurrence of these cases. It is the œsophagus of a new-born baby—I suppose only a few days old, because it must certainly have died of its malformation. The œsophagus is well developed in the upper half; the lower half is complete but very narrow, and it comes out of the trachea instead of being attached to the upper half of the œsophagus. There are other specimens in which neither end of the œsophagus—neither of the upper nor lower segment—opens into the trachea, but in which the œsophagus is replaced between the upper and lower segment by a fibrous cord. But you can imagine perfectly well a separation not so complete as this, in which the upper and lower segments of the œsophagus are fused together, but so indifferently that a constriction exists at the seat of fusion, a kind of diaphragm with a hole in the centre, which would account for the condition in the two patients I have described. Of course, when I speak of people under the age of eighteen or twenty, and how rare it is to find chronic difficulty of swallowing, I am excluding those very rare cases indeed in which a patient has swallowed some caustic, such as caustic soda or caustic potash, and has a fibrous or cicatricial stricture.

Then I come to the next period, which I shall take as from twenty to forty years of age. And again I should say that chronic difficulty in swallowing is not by any means common during that period. In the first place, malignant disease of the œsophagus is very rare before forty. And, although you might think from what you read in books that innocent stricture of a fibrous or cicatricial character is quite an ordinary thing, all I can say is that in my experience—and I have had a very considerable experience of diseases of the œsophagus—I do not see one case of innocent stricture in a year, and I do not think I have seen one case in the last three years. So I feel sure they are very rare. So if you exclude spasmodic stricture of the œsophagus, your mind will turn, if you are accustomed to chronic cases of dysphagia, to the probability of difficulty in swallowing in connection with the larynx, perhaps with the back wall of the larynx, the cricoid plate. Here is a specimen of malignant disease of the cricoid plate, and here is a specimen of tubercular disease of the larynx. Those are the two conditions to which I should first look for. an explanation, malignant disease and tubercular disease, particularly about the arytenoid region and interarytenoid fold, aryepiglottic region and cricoid plate. And I cannot give you a better example than that poor young woman who is in Paget Ward at the present time, and on whom I operated yesterday in the hope of being able to remove the disease. Some two or three weeks ago I was asked to see her at a nursing home, on behalf of a lady who was very much interested in her. She is twenty-eight years of age, and she tells me she has had difficulty in swallowing for a very few weeks; but she has not lost flesh, and her difficulty has been, she says, very much less during the last few days. She also states that she can take her food quite nicely compared with what she could do a few days previously. So I had it in my mind that quite probably she might be suffering from muscular spasm, spasmodic stricture of the œsophagus; but the circumstance that made me doubtful about her was that she always pointed to the same spot as the seat of her trouble, which was at the level of the larynx. More than that, she said that, although she had improved very much in swallowing, she still could not take any solid food except the softest. I examined her neck very carefully, and, before passing an œsophageal bougie, I examined her with the laryngoscope, and to my very great disappointment discovered a raised warty area on the wall of the pharynx, above the level of the larynx. If you have once seen a condition of that kind, such an appearance makes you suspicious and almost certain of the existence of epithelioma about the cricoid plate. And if you think that it is too rare a disease for a person of her age, because it is reported to be so, I can tell you of other cases in younger people than she is. I remember, for instance, being called in to see the daughter of one of the tradesmen in the Strand some years ago. I was told she had had difficulty in swallowing for some months, and that she had wasted very considerably. Her voice was very feeble, I was not sure whether from general weakness or on account of the existence of actual disease. On examining her I discovered, at the upper part of the cricoid plate, a warty growth, and there were enlarged glands on both sides of the neck. There could be no doubt she was suffering from cancer. And then, to my very great astonishment—for I had never seen such a case in a patient so young,—I learnt she was only

Digitized by Google

twenty-four. On hearing that the treatment could not be radical and that I could not give any hope of permanent success, she declined any palliative operation, and died in the course of a very few weeks. So that if the age of the patient be between twenty and five and thirty years, although you may have in mind the probability of tuberculous disease, you must not exclude the possibility of the existence of malignant disease.

Now I come to the age after forty years, when difficulty of swallowing, chronic dysphagia, becomes very much more frequent. And in my experience by far the most common cause of it is malignant disease of the œsophagus. If a man of more than forty years of age is brought to my house, or is sent to me with a history of difficulty of swallowing referred to the gullet, the first thing I have in my mind as a likely cause is malignant disease. And here, again, personal experience is far more valuable than mere book-work. If you take a book on diseases of the throat or on diseases of the œsophagus—such a book, for instance, as that by the late Sir Morrell Mackenzie,—you will find so many diseases which may produce chronic dysphagia in persons over forty years of age that you may be confused. Some patients have dilatation of the œsophagus, some have innocent strictures, some have diverticula, and some suffer from central or peripheral nervous disease. But all those things are rare. For every one of this kind of case you will probably see nine cases of malignant disease of the œsophagus. But on the other hand, as there is a possibility of one or other of these different conditions being present, it is very important not to overlook them and come instantly to the conclusion that the patient, on account of his age and symptoms, must be suffering from malignant disease of the œsophagus. You must remember, too, that malignant disease in any part, from the mouth right down to the cardiac end of the œsophagus, may give rise to difficulty in swallowing. As all kinds of malignant disease are very much more common after forty years of age, particularly in the mouths of men, you must be on the look-out for malignant disease of every part, from the base of the tongue particularly, where it is not by any means uncommon, down to the cardiac orifice of the stomach.

Next with regard to *sex*. Men are more liable to chronic difficulty in swallowing than women are. And in men the cause of difficulty in swallowing is, most frequently, malignant disease. In women it may be the same, but in women, particularly young women, you may meet with nervous difficulty. So that in men of a certain age you look chiefly for malignant disease. In women also you look for malignant disease, but you are on your guard lest the case should be one merely of nervous spasm.

I now come to another very important point, *the inconstancy of symptoms*, particularly in the ability to take various kinds of food. I suppose ninety-five out of every hundred persons with chronic dysphagia can take fluids, as you would naturally expect, very much better than they can take solids; and if a person, particularly if it be a young patient, is brought to you with chronic difficulty of swallowing, and the history is that solid food is very much more readily taken than liquid food, the chances are that the trouble is nervous, that it is a spasmodic or nervous stricture. But there are certain exceptions to the rule. For instance, in cases of disease about the cricoid plate of the kind of which I have already spoken, fluids are apt to produce spasm, with all that choking and distress which occurs when food "passes the wrong way," so that soft solid food, such as jelly and blanc-mange, is often better swallowed than fluids. You must be on your guard against being too much influenced by what the patient tells you about being able to take this or that food better than another. You will find patients exaggerate their ability to take certain kinds of food, and they do that partly in order to gain a more favourable opinion from you, and partly, I am sure, to place their case in a more favourable aspect before themselves. For instance, a gentleman with malignant stricture of the œsophagus, for whom I performed gastrostomy on account of increasing difficulty in swallowing even liquids, told me that he ate walnuts every night, though I knew he could not take his ordinary dinner. I have no doubt he chewed them very fine indeed, but he did take them, and kept them down, because his wife agreed that no part of these walnuts ever came up. He told me this with very great satisfaction, because to him it was a kind of pledge that he was not suffering from any very serious condition, and he looked upon his trouble as in all probability due to some kind of nervousness and not to organic disease, as far as he under-

Digitized by Google

stood these things. But I am sorry to say that in spite of this, within a few months of the performance of the gastrostomy he died of his malignant disease.

Again, there is the question of the *reputed seat of a stricture.* I must warn you that the patient, although in many instances he is able to give you a very good idea of where the stricture is, may be absolutely unable to do so, and may even mislead you. The referred sensations in connection with troubles about the throat and upper alimentary canal are so singular and uncertain that the most curious mistakes may arise. I remember a friend of mine, a noted laryngologist, telling me that he was staying in a country house, and that there was staying in the same house at the same time another distinguished laryngologist. My friend one day got a piece of bone stuck in his throat. He was in too great pain to continue his dinner. He went up to his room and with his finger tried to find and remove the bone, but could not do so. Presently up comes the other professor, saying, "What is the matter?" "Oh," he says, "I am in great pain; I have a piece of bone in my throat." Between them they had a laryngoscope, and the one professor looked down the other professor's throat and began to smile. "What are you smiling at?" asked my friend. "Where did you say it is?" "It is down here, on the right side about the larynx." "No," he said, "it is high up, on the left side behind the palate." And from there he took it out. This anecdote shows how a patient may be mistaken not only as to the level of organic disease, but even as to the side on which it lies. My own impression is that, as a rule, patients place the seat of stricture at a higher point than it really is, except in connection with disease at the back of the larynx, in which case they are generally quite right as to the seat of stricture. I have noticed many times that people with malignant disease at the cardiac orifice of the stomach believe the seat of stricture to be much higher up.

I have spoken hitherto of dysphagia, difficulty in swallowing. And you must know, of course, that there is another "phagia," that is odynphagia, in which the patient suffers pain in swallowing. A patient may have dysphagia without odynphagia, and he may have pain without much difficulty in swallowing. He may have pain in swallowing, but he may be able to swallow food, every kind and any kind. For instance, non-malignant strictures of the œsophagus, as a rule, do not give rise to pain in swallowing; and even malignant strictures of the œsophagus, unless very high placed, may exist for months, or more than a year, and yet never at any time cause the slightest pain in swallowing. On the other hand, a spasmodic stricture, which you would think ought not to be associated with any pain, sometimes causes very great pain. I take it that when the bolus of food attempts to pass, or is carried down to the point where the œsophagus grasps it, it does so with such force that a pain like a very severe cramp is produced. You know what the pain of cramp in the leg is, and you can fancy what the pain of spasm of the muscular coat of the œsophagus may be; probably that accounts for the extreme pain from which some of these people suffer.

Now I have to point out to you that difficulty in swallowing may be due not to any trouble *inside* the alimentary canal between the mouth and stomach, but may be due to *external* trouble,—that is, to something pressing upon the œsophagus. There may be enlarged glands, or a tumour of the thyroid gland, particularly malignant disease of the gland which presses upon the œsophagus and causes difficulty in swallowing. There may be an aneurysm of the aorta; and for a long time it may be very doubtful what the difficulty in swallowing depends upon. I have lately had such a case in a gentleman between fifty and sixty years of age, whom I have examined from time to time during more than a year. He is a very gouty subject, and about one and a half or two years ago, when he was recovering from a very severe attack of gouty psoriasis and eczema, he was attacked for the first time with some difficulty in swallowing. As the psoriasis cleared up, his difficulty in swallowing became more troublesome, and he consulted a surgeon in a neighbouring town, who, after a time, sent him up to me. I found that he could apparently swallow well on some occasions, and that on other occasions he had very considerable difficulty, which was associated with the bringing up of a quantity of mucus. His chest had been examined very carefully indeed, and there was no physical sign of trouble there. Not being satisfied as to the nature of his complaint, I passed a bougie, but not easily. Believing he might be suffering from gouty spasm, I had him placed under an anæsthetic, and I passed

Digitized by Google

a bougie into his stomach, a bougie of considerable size. I was conscious, after it had passed the cricoid cartilage, that it did not go down quite as smoothly as I thought it ought to do; and I wondered whether he had a condition of the mucous membrane of the œsophagus similar to that leucomatous or leucoplakic condition which you find in the interior of the mouth. I advised that a bougie should be passed at regular intervals, and this was done for some time. In the summer he went out fishing and appeared to have caught cold after standing about a long time, and then he had an attack of acute difficulty in swallowing. I saw him again about July, and there was no development of his symptoms which would tell us what was the matter with him. It was suggested to me that it might be a case of œsophageal pouch; but I absolutely negatived that, because he did not bring back any portions of undigested food. I have seen him again in the course of the last few days, in consultation with a physician. He now has paralysis of the left vocal cord; he has dyspnœa; he is sometimes able to swallow perfectly well, at other times badly; he has wasted very much, and there is no doubt in the mind of the physician and myself that he is suffering from aneurysm of the aorta. We do not think the aneurysm is the direct cause of his dysphagia; for a large aneurysm of the arch of the aorta may exist without producing marked dysphagia. We think that this gentleman's difficulty is due not so much to direct pressure of the aneurysm as to the interference with the nerve supply and to reflex spasm in a nervous gouty man, caused by the presence of the aneurysm. We judge of that by the symptoms, and by the fact that, although he can often swallow well, every now and then he is absolutely unable to swallow any kind of food, and has to be fed by the bowel for two or three days at a time.

From what I have said you will see that it is of the greatest importance in a case of chronic difficulty in swallowing to examine every part, both from the inside and the outside; and to make a most methodical and careful study of every patient who is brought to you, because there is more room for mistake in connection with this kind of trouble than in connection with most others. For my own part, I examine first the outside of the throat and neck, and the chest, so as to be sure that there is no palpable tumour or undue fulness of the veins.

I particularly examine the posterior triangle of each side, where the presence of small shotty glands may indicate malignant disease very much lower down. I examine the interior of the mouth; every part of it, from the opening of the mouth right down to the larynx; and I examine the interior of the larynx with the laryngoscope. I particularly see whether there is any disease on either side of the throat above the larynx; whether there is swelling or ulceration of the epiglottis or posterior part of the larynx; and I look also with the greatest care to be sure that the vocal cords move readily. Paralysis of one vocal cord is a very important symptom in a case of this kind; it means generally one of two things: malignant disease of the œsophagus or an aneurysm of the aorta. Paralysis or paresis of both vocal cords may mean disease of the central nervous system. I make all these examinations with the greatest care before I pass an œsophageal bougie. I ask, too, about the presence or absence of cough of a spasmodic character; about difficulty in breathing, or vertigo; whether there is expectoration, and if so, of what character it is; and I note the character and quality of the voice of the patient. Also I look for indications of disease of the nervous system, particularly of bulbar disease. If, in a case of supposed stricture of the œsophagus, you begin by passing a bougie, you may meet with a terrible disaster. The supposed stricture may be due to the presence of an aneurysm of the aorta; and if the aneurysm is making its way into and threatening to open into the œsophagus, the thin wall may be ruptured by the bougie, and the patient may die upon the place. A careful examination of the history and symptoms of the patient will almost always serve to warn you that the passage of a bougie is fraught with danger.

There is one very important aid to diagnosis in these cases, and that is the use of an anæsthetic. If there is doubt whether œsophageal obstruction is due to the presence of organic disease, the patient should be persuaded to allow the bougie to be passed under an anæsthetic. When the muscular reflex has been set at rest by chloroform, it is generally quite easy to determine the existence, the seat, and the degree of real constriction. Owing to neglect of this precaution, I remember well, to use a vulgar expression, having my eye wiped some years ago by one of my friends. Both he and I saw an old lady about seventy years of age;

Digitized by Google

I examined her without an anæsthetic, and so did he. But he had the good sense afterwards to examine her with an anæsthetic, and he then passed an instrument easily into the stomach, whereas the instrument I had passed without an anæsthetic stopped at the cardiac orifice. He came to me very pleased, and said, "Butlin, I have wiped your eye;" and it was perfectly true. On the other hand, there are cases in which serious disease, even malignant disease, of the œsophagus exists, and there is dysphagia, yet a large bougie can be passed into the stomach under an anæsthetic. The disease, without producing gross constriction or forming a ring in the wall of the œsophagus, which would hinder the passage of a bougie, interferes with the function of the œsophagus, and so causes difficulty in swallowing.

Finally, I would have you very cautious in offering a diagnosis and prognosis of any case of chronic difficulty in swallowing in which the disease is beyond the reach of sight and touch. The evidence is for the most part circumstantial. I believe I am not far outside the truth when I say that nine of every ten persons who are brought to me with dysphagia, apparently of œsophageal origin, are suffering from malignant disease of the œsophagus; and the diagnosis is generally not difficult. Indeed, one is far too seldom wrong in the sad prognosis of the case. But, as the disease is beyond the reach of the finger, and out of sight, it is always well to speak guardedly. There are patients who do not present the usual symptoms of malignant disease of the œsophagus, and in whom a bougie can be passed with ease into the stomach, who nevertheless die, and the disease is found to be malignant. And there are other patients who present the most marked symptoms of cancer of the œsophagus, and who are condemned to death by every specialist whom they consult, and who nevertheless mend and completely recover. Quite recently, a gentleman in whom neither I nor other medical men who saw him could find evidence of organic stricture of the œsophagus, and in whom a bougie could apparently be easily passed into the stomach, has died (in spite of successful gastrostomy) of wasting and exhaustion quite characteristic of malignant disease. On the other hand, I remember well a case of the other kind, of a publican who was brought to me with chronic dysphagia. He had wasted rapidly, could only swallow liquids, and brought up large quantities of frothy mucus. A bougie was arrested low down in the œsophagus. When his doctor, who came with him, talked the case over with me, I said, "He will not live six months." He said, "Will he require gastrostomy?" To which I replied, "He may do, but I should not recommend the operation unless he is really in danger of starvation." For, at that time, I did not care to perform gastrostomy if the patient could get down sufficient nourishment to allay the pain of hunger. Many months later I met the doctor, and I asked, "When did that poor fellow die?" "Oh," he replied, "he did not die. After a while he began to grow better, and has now completely recovered."

So, too, recovered a gentleman who was sent to consult me from Hants. He had seen many medical men and several specialists, and they were all agreed that the obstruction which they found at the lower end of the œsophagus, and the wasting and other symptoms of the patient, could only mean cancer. I thought they were probably in the right. But, as there were certain curious features in his case, I, warned by the recovery of the publican, gave a less decided diagnosis, and held out a faint hope of a happier issue of the illness. I never heard of him for four or five years, when his medical man told me that he was so completely recovered that only a few nights previously he had met him at dinner, where he ate and drank like all the other guests, and was as well nourished and as hearty as ever he had been.

The œsophagoscope, which we are just beginning to use, may help us in some of these doubtful cases. But, until you can use it with confidence, let me warn you to speak with caution of the cause and future of every case of chronic dysphagia in which you can neither see nor feel the disease.

THE combined action of butyl-chloral hydrate and gelsemine is taken advantage of for the relief of acute neuralgia of the fifth nerve, and a "Tabloid" product is now issued by Burroughs Wellcome & Co. which provides a convenient means of securing the desired therapeutic effect. The formula is:—Butyl-Chloral Hydrate, gr. 3; Gelsemine Hydrochloride, gr. $\frac{1}{200}$. In view of the powerful action of gelsemine hydrochloride it is necessary that the most precise instructions be written by the medical man for the administration of this product.

Digitized by Google

A LECTURE ON NASAL POLYPUS.

Delivered at the Polyclinic.

By H. LAMBERT LACK, F.R.C.S.,
Surgeon to the Throat Department, London Hospital.

GENTLEMEN,—The subject upon which I wish to speak to you to-day is as old as any subject in medicine, but has recently excited a good deal of attention. I shall try to give you a short account of the modern original work which has been done on it, as well as of my own researches.

Nasal polypi may be defined as new formations springing from the ethmoidal region of the nose, consisting mainly of a loose fibrous network and containing more or less of the other tissues normal to the region. They are roundish or pyriform growths, often pedunculated, pinkish or bluish in colour, semi-translucent, and varying in size from a currant to an acorn, or larger. They were formerly regarded as tumours, either myxomata, adenomata, or soft fibromata. In more recent times it has been recognised that they are inflammatory in origin, although the cause of the inflammation was unknown. Among other things it has been stated that polypi may arise from the irritation produced by foreign bodies in the nose, by malignant growths, by sinus suppuration, syphilis, etc., or by any form of severe chronic rhinitis. Woakes stated that polypi were merely symptoms of a bone disease which he called necrosing ethmoiditis, but his views acquired practically no support. I shall endeavour to show that nasal polypi should be regarded as simple localised patches of œdematous mucous membrane, and that this œdema depends upon a rarefying osteitis of the underlying bone. Before discussing these theories, however, I should like to draw your attention to some of the chief pathological and clinical features of the affection.

1. Polypi are commonly bilateral, and five or six or more growths may be met with in each nostril. Occasionally a single growth is found, which then usually attains a very large size. It is often stated in books that as many as fifty or sixty or even more polypi may be found in a single nostril. This is evidently an error of observation, due regard not having been paid to the fact that polypi recur very rapidly. No such number of growths could ever be removed from a nose at a single sitting, nor have they ever been seen on post-mortem examination.

2. *Method of attachment.*—Polypi may be pedunculated, with a more or less narrow peduncle, or sessile. The two forms are not uncommonly associated, and all varieties exist between the pear-shaped polypus with a long narrow peduncle and the broadly attached sessile growth. Again, we may find a thickened patch of mucous membrane, more or less imperfectly divided from the surrounding mucous membrane; and it is impossible to decide whether we should call it a polypus, or merely an œdematous area. The difference is entirely one of degree, the one shading into the other. If the mass is sufficiently pronounced to be movable with the probe, it is looked upon as a sessile polypus; in a less degree, it is considered simply a thickening or localised œdema of the mucous membrane.

3. Polypi are most commonly met with in early adult life. They are rare under the age of fifteen, and especially so under the age of ten. This has an important bearing upon the ætiology of the growths, for in children the accessory sinuses are not developed, neither is the ethmoid bone.

4. *Frequency.*—In a special clinic nasal polypi are met with in about one in twenty cases. Post mortem they are apparently met with more often, and Zuckerkandl says that he has found nasal polypus in one in every eight or nine examinations.

5. *Point of origin.*—Clinically, nasal polypi appear to grow from the middle turbinate and from the underlying parts, but their origin is often deeper than is suspected. As the result of post-mortem examination, polypi have been found to spring most frequently from the uncinate process, the bulla ethmoidalis, the infundibulum, and from the lips of the ostia of the various cavities, the antrum, and the anterior ethmoidal cells. Next most frequently they spring from the middle turbinate, and very rarely they may be found attached to the superior turbinate or walls of the posterior ethmoidal cells, the cribriform plate, and the upper part of the septum. Thus it is seen that in every case polypi are attached to some part of the ethmoid bone, most commonly that forming the upper part of the middle meatus.

6. *Liability to recurrence.*—The great tendency of nasal polypi to recur after removal is so con-

Digitized by Google

stantly observed and widely recognised that it need not be insisted on. I have had one case in which polypi were removed every fortnight for three years, and yet during all that time the nose was never clear. In other cases polypi have been removed from the nose at intervals during ten, fifteen, and even twenty years, and such cases are by no means rare. Of course cases are met with —usually those in which a few polypi are present —in which no recurrence takes place after removal, but this happy event is by no means usual. Recurrence must be regarded as the rule, and cure after one or two removals as the exception.

7. *Histological structure.*—On microscopical examination polypi are found to consist of a loose fibrous network, the meshes of which are filled with serous fluid. This fluid does not contain mucin, as was formerly considered. In parts the fibrous stroma may have undergone more or less myxomatous degeneration, but a purely myxomatous growth is never found. The growth is covered by an epithelial investment. In the older growths this investment consists of several layers of epithelial cells, the most superficial of which is columnar and ciliated. In the more recent and rapidly growing polypi the epithelial investment gradually becomes thinned out at a distance from the attachment of the growth until, when the apex of the polypus is reached, it may consist of a single layer of cells, most usually cubical, or in places it may even be deficient. In addition to these elements, polypi contain a certain amount of all the other tissues normal to the region—the glands, vessels, nerves, etc. The glands are especially numerous in the sessile and slower-growing polypi. They may be normal in character, or they may be dilated so as to form cysts. These cysts are almost always met with in polypi, and are specially large in the growths which occur far back in the nose. They are caused by dilatation of the acinus of a gland as the result of obstruction of its duct by pressure of inflammatory exudation or by contraction of new-formed fibrous tissue. Scattered throughout the growth are masses of round-cells. These are especially marked around the vessels and glands and in the more rapidly growing polypi.

To sum up, then, histologically polypi are found to consist of all the structures normal to the part from which they grow *plus* a certain amount of inflammatory exudation—serum and round-cells.

8. *Relationship to sinus suppuration.*—This subject has attracted attention, chiefly owing to the assertion of Grünwald that polypi may be considered as pathognomonic of sinus suppuration. In a series of fifty-three cases Grünwald demonstrated sinus suppuration in thirty-eight. His observations have been frequently tested by others, and have not been corroborated. We may dismiss the older observations, owing to the fact that our means of diagnosis of sinus suppuration have until quite recently been very imperfect, and the frequency of sinus affections has been greatly underestimated. Granting this, however, and admitting that when any purulent secretion is met with in the nose a sinus suppuration is probable, it seems that sinus suppuration may be excluded in all but about 30 per cent. of cases of polypi. Of course, in a few cases sinus suppuration may have been present, and have subsided before the case came under observation; but in view of the fact that sinus suppuration is met with in only a third of the cases of polypi, it is impossible to say that polypi depend upon it. This subject will be again alluded to.

9. *The existence of disease in the underlying bone.*—So far I have not alluded to the changes in the bone. Woakes stated that polypi were merely a symptom of a bone disease which commenced as a fibrosis and went on to obliteration of arteries and the absorption or necrosis of the bone, with the formation of polypi. His views were based partly upon clinical observation, and it was quickly pointed out that the methods which he employed were faulty, and this prevented the acceptance of his views. On the other hand, pieces of bone removed by him were submitted to microscopical examination by Thurston and Martin, by whom the existence of definite bone changes in a considerable proportion of cases was demonstrated. In 1898 and 1899 I devoted special attention to this subject, obtaining several pieces of bone from over thirty cases of nasal polypi. These pieces were all submitted to microscopical examination, and all showed various degrees of osteitis. The first change, apparently, was a thickening of the deeper layer of the periosteum, which became changed into two or three layers of large nucleated cells. The edges of the bone near the periosteum

Digitized by Google

seemed to become more cellular than usual from increase in the size and number of the bone cells. Then indentations formed along the edges of the bone, and in these little cup-shaped depressions were seen large nucleated or multinucleated cells. In this way cavities were hollowed out in the bone, and this process gradually progressed until the bone was broken up. In specimens in which the changes were more advanced, larger and smaller fragments of bone would be seen entirely surrounded by cells, and obviously in process of complete absorption. These appearances indicate a rarefying osteitis. True necrosis was never observed. In a few cases, in which single polypi of long standing were met with in the nose, and in which a piece of bone was removed, another condition was demonstrated. The bone, when cut through with the snare, was found to be unusually hard and dense, and this sclerosis of the bone was confirmed by microscopical examination. In such cases there is no tendency to recurrence of the growth, and it seems fair to assume that when, from any reason, spontaneous cure takes place, the rarefying osteitis ceases, and results in a sclerosis of the bone, and that this is the natural method of cure.

The existence of these extensive bone changes does not rest upon microscopical evidence alone. The following clinical evidence may be adduced:—When, for any reason, a general anæsthetic was administered, the finger was passed well up into the nose and the ethmoidal region explored. In such cases the existence of broken-up spicules of bone could often be readily determined, and sometimes rough bare bone could be felt. A similar result may be obtained by examination with the probe, but the soft jelly-like mucous membrane is so easily penetrated that it is necessary to use a blunt-pointed probe, and make the examination with the greatest care and gentleness. If the sharp-pointed probe recommended by Woakes be used, bare bone can be detected in the normal nose.

Another piece of evidence may sometimes be obtained if a case of extensive polypi be seen in which no operation has been performed. If the polypi be removed one by one with the snare, without touching the bone, it will be found that the middle turbinate has completely disappeared. Again, as already stated, in a few cases, on snaring off the anterior end of the middle turbinate, definite sclerosis of the bone, as determined by its hardness and density, is found. This is in accordance with the statement of Zuckerkandl, who said that he had never found osteitis in cases of polypi, but had occasionally met with sclerosis. His statement was based upon the examination of two or three cases met with post-mortem, in which it was probable that all the acute changes had subsided.

Ætiology.—It is now possible, in the light of the above information, to discuss the various theories as to the cause of nasal polypi.

The idea that nasal polypi are tumours in the strict sense of the word can be at once dismissed when it is remembered that nasal polypi consist of all the structures of the nasal mucous membrane, that they pass into it at their edges without any definite line of demarcation, and that, although obviously simple tumours, they have a tendency to the most marked and rapid recurrence. Both macro- and microscopically all varieties of growth may be met with between simple swelling or œdema of the mucous membrane and sessile and pedunculated polypus. Moreover, as Grünwald showed, by tightly packing the maxillary antrum, œdema of the lower lip of the maxillary ostium can be produced within twenty-four hours, and this œdematous swelling is indistinguishable, either by the naked eye or by the microscope, from a true nasal polypus.

The theory that nasal polypi are pathognomonic of sinus suppuration must also fall to the ground. As we have seen, nasal suppuration is not present in a large majority of cases. It is true that it is present in a considerable number, and the connection between the two affections is not quite obvious. It is possible that in some cases sinus suppuration leads to inflammation of the underlying bone and thus to polypus, and that in other cases the original acute inflammation produced both the sinus suppuration and the osteitis simultaneously.

The theory that polypi are ordinary granulations, peculiarly modified by the conditions under which they grow in the nose, is also opposed to facts. Granulations in the nose, such as are met with in connection with necrosis, the presence of foreign bodies, and after certain operations upon the septum, are similar to granulations elsewhere—a little more exuberant perhaps. They consist of round-cells, spindle-cells, and new-formed fibrous

Digitized by Google

tissue, with capillary loops. They contain no glands or other specialised structures; they are not covered by a layer of surface epithelium. To the naked eye they are pink, and readily bleed on touching. They disappear when their original cause is removed.

We are now left with the option of either agreeing with Morell Mackenzie that the cause of polypi remains unknown, or of adopting the theory which I have put forward, and which was first advanced by Woakes, that polypi depend upon osteitis in the underlying bone. As we have seen, bone disease is a constant concomitant of nasal polypi; its presence can be demonstrated both by clinical and microscopical examination. It remains to be considered whether the bone disease is the cause of the polypi, or—as is frequently asserted—polypi are the cause of the bone disease. This point may be decided by the results of operation. If a severe case of nasal polypus is treated by removing the polypi, recurrence is certain. If, on the other hand, the bone also is removed in the way about to be described, recurrence is a rare event. The beneficial result of removing a layer of bone with a polypus was noted by Morell Mackenzie, who states in his work that in two cases in which recurrence of nasal polypus had rapidly taken place, a cure was thus produced. The same procedure had been previously recommended by Pirogoff and Sir William Ferguson. In face of these results I think it must be maintained that polypi are the result, and not the cause, of the disease in the bone.

One other word may be added here. Although it seems certain that polypi, as usually met with, are due solely to disease in the underlying bone, still as polypi are simply œdematous infiltrations of the nasal mucous membrane, it is obvious that in some cases other causes may produce similar effects. Thus Grünwald produced what looked like a polypus by tightly packing the maxillary antrum, and in some cases it is probable that the œdema of the uncinate process—so common in antral suppuration—may proliferate until it forms a polypus. In malignant disease of the nose also, as is well known, polypi are commonly met with, and may be looked upon, as Luc suggests, as a neighbouring œdema.

When we go further, and try to discover the cause of the osteitis, we are on more uncertain ground. It is not tuberculous, syphilitic, or traumatic. We must remember, however, that polypi grow only from the ethmoidal region of the nose, and that in this region the connection between the mucous membrane and the bone is a very intimate one. The mucous membrane is thin, and the periosteum is so intimately blended with it that any acute or severe catarrh of the upper part of the nose must affect the periosteum as well as the mucous membrane. Thus it is probable that any acute catarrh of the nose, especially if severe or prolonged, may produce periostitis and osteitis. It seems probable that polypi have been much more common in recent years, and that this frequency must be in large part ascribed to the prevalence of influenza.

The symptoms and diagnosis of nasal polypi are too well known to be discussed, and I shall therefore at once pass on to the subject of treatment.

Treatment.—In considering the treatment, the relationship between nasal polypi and the underlying bone disease must be carefully borne in mind. The following types of cases can, I think, be easily defined:

1. Those in which one or two polypi only are present which are of long standing, as shown by the history, by the large size and the pedunculated nature of the growths. In these cases the snare may be employed, although it is well to pass the loop well up round the base of the growth, and to include a piece of bone whenever possible. In such cases it is probable that the active bone disease has passed off, or at any rate is not very extensive, and thus the operation with the snare is likely to be successful.

2. The second group will include cases of limited disease, when polypi are found growing from the middle turbinate or from the anterior part of the middle meatus only. Here also the best procedure is to remove the growths with the snare under cocaine anæsthesia, and later if there be any tendency to recurrence, to cut away the diseased bone with punch forceps. In some cases Grünwald's forceps alone will effect this; in others Hajek's hook will be found a very convenient aid. In many cases it is necessary to remove the anterior end, or more, of the middle turbinate, even if not affected, so as to gain access to the diseased area. Should this treatment not be effectual, a small operation may be performed under gas. The patient is seated upright in the usual rhinoscopic position,

Digitized by Google

and, under direct illumination, a ring knife is passed up over the affected area and the disease rapidly scraped away.

3. We now come to a third group, in which extensive disease is present. In this group I should include all cases in which the nasal fossa seems full of polypus, or in which extensive polypoid degeneration of the ethmoidal region exists; cases in which repeated recurrence has occurred after simple operations, and especially when the above conditions are complicated with sinus suppuration. In these cases I consider it best to give the patient a general anæsthetic, and to scrape away the whole of the polypi and diseased membrane and bone. A few points about the operation may be mentioned. I prefer the patient lying on his side in the horizontal position. The instruments required are an ordinary pair of nasal polypus forceps to remove a few of the larger growths, and one or two sharp curettes. The curettes that I use are Meyer's original adenoid curettes or ring knives, which are much more effective than any sharp spoon. Care must be taken in introducing the polypus forceps not to push them through the cribriform plate, an accident which has occurred. When introducing the ring knife, also, it is better to introduce it with its blunt surface towards the septum and the sharp-cutting edge directed outwards towards the orbit, and when scraping all force should be used in this direction. The curettement is continued until all soft bone and mucous membrane is removed, and until the edge of the knife comes down on to firm bone, upon which it will take no hold. Having done as much as possible with the large knife, a smaller size may be introduced, and used with even greater care until every trace of disease is removed. The hæmorrhage is often sharp, and for this reason the anæsthetic must not be pushed too deeply, and the patient must be rolled well over on to his side, so that all blood may escape from the nose. As a rule the bleeding soon ceases, especially if the face be douched with cold water. If not it may be necessary to pack the nose, but this should be avoided whenever possible. The after-treatment consists in simply cleansing the nose with an alkaline or mildly antiseptic lotion, such as boracic acid. If all the growths be removed—and with a little practice this is usually possible—the patient is cured with a single operation. The upper part of the nose heals up into a dry cavity, lined by pale, closely adherent mucous membrane. This gives rise to no subsequent trouble and does not produce dryness of the throat, as may occur in operations on the inferior turbinate.

The dangers of this operation are obvious, but may be briefly alluded to. The region of the cribriform plate must be carefully avoided by directing the blade of the knife outwards, as above directed, and by care in introducing the polypus forceps. The orbit, on the other hand, may be entered with impunity. Sometimes a black eye follows the operation, but soon subsides. In one case only—a severe case of ethmoidal suppuration—did an orbital abscess follow some two months later, and require to be opened. In a few cases rises of temperature, headaches, etc., have occurred. I have operated now in over 100 cases, and there has been no fatal result. This shows that the operation—if performed with care and proper precautions—is not very dangerous. On the other hand, it presents practically the only chance of a cure in the most severe cases, and therefore its performance is fully justified in all cases in which extensive polypi are present, especially when associated with suppuration in the ethmoidal cells, and when repeated recurrence has taken place after simpler methods of removal. The most important contra-indication to the operation is the age of the patient. As age increases the ethmoid becomes more brittle, and I would hesitate to operate on patients over forty years of age, unless for very exceptional circumstances.

Should this operation not be carried out, there are only two alternatives. One is to leave the disease alone, to be content to remove the growths as they recur, and thus endeavour to keep a free passage through the nose. The other is to attempt to remove the diseased bone piecemeal by means of repeated small operations. This—the generally recommended method—consists in cocaining the nose and breaking away a small piece of bone with forceps or hooks. Twenty, thirty, or more operations are required, and even then very little can be done. On the other hand, the operations are always painful, as cocaine acts but feebly in these cases. The patient usually wearies of the treatment, and abandons it before any improvement has been obtained. Also the idea that these operations can be performed under direct illumination

Digitized by Google

is a delusion. Directly the mucous membrane is touched, bleeding takes place and obscures the view; and a much better idea can be obtained by carefully observing the position of the patient and keeping in mind the anatomy of the parts when the patient is quiet under a general anæsthetic, than when an attempt is made to operate with reflected light. It is probable even that these repeated operations are more dangerous in the aggregate than a single more severe one, and the results cannot be compared.

In conclusion, therefore, the views that I wish to urge are two: first, that the ordinary nasal polypus is merely a local œdematous infiltration of the mucous membrane, and is dependent upon disease in the underlying bone; and secondly, that the treatment consists in removing not only the polypus, but in dealing thoroughly with the bone itself.

WITH MR. D'ARCY POWER
IN THE OUT-PATIENTS' DEPARTMENT OF
ST. BARTHOLOMEW'S HOSPITAL.

July 20th and 27th, 1903.

Gastro-enterostomy.

This man, æt. 50, swallowed about an ounce and a half of strong nitric acid on New Year's Eve, 1902. He vomited repeatedly, and was taken to the German Hospital, where he remained for eight days, and was discharged in apparently good health and without any difficulty in swallowing. On January 25th he vomited after eating a hearty dinner of pork, the act of vomiting being preceded by great pain in the epigastrium. From this date until his admission to the hospital in March he vomited daily, and often twice a day, always suffering great pain. The pain was referred to the epigastrium, and gradually increased until it attained a maximum about two hours after food. The vomiting at first used to occur directly after eating, but the length of time increased gradually until on admission there was usually an interval of two or three hours. No bile was ever vomited. On admission to the hospital the patient was found to be a spare and constipated man, with a dilated stomach and thickened pylorus.

I performed a precolic gastrostomy upon him on March 9th. The stomach was dilated, congested, and markedly thickened in the neighbourhood of the pylorus. The anterior wall of the stomach was made to anastomose with the jejunum about nine inches beyond the duodeno-jejunal angle, but without cutting away any of the mucous membrane either in the stomach or jejunum. The aperture between the stomach and the jejunum was purposely made small, and the incision in each viscus was not more than half an inch in length. The whole operation lasted forty minutes, and the patient bore the operation well, his pulse at the end of the operation being 96. On the following day it was reported that he had not vomited, his pulse was 76, and temperature was 99° F. He made an uneventful recovery, and was discharged on April 22nd free from pain and without any return of vomiting.

Here, as in other abdominal cases, the method of feeding is of great importance, so I will tell you what he had. Nothing was given him by the mouth on March 10th, the day following the operation, except an occasional drachm of water. On March 11th he was ordered a drachm of whey occasionally, and on the 12th this amount was increased to two drachms, with instructions to the nurse that the quantity might be increased to half an ounce every hour if he were not sick. On March 13th he had half an ounce of milk and half an ounce of water hourly, to be increased to three ounces every two hours unless he was sick. On March 14th he took five ounces of milk every two hours, with two feeds of beef essence in the course of the day; and he received the same quantity of food until March 16th, when the amount of essence was increased to half a pint in the twenty-four hours. On March 17th I added two ounces of brandy to his dietary, which was further supplemented by two eggs on the 19th. He was placed upon the hospital half diet with two eggs and two ounces of brandy on March 21st, and this was continued until April 7th, when he was ordered full diet. He complained of some pain in his stomach after food on April 15th, but this was promptly relieved by cutting off his potatoes and bread and giving him minced meat.

You know that there are several methods of performing the operation of gastro-enterostomy, and you have seen me do them all at various times.

Digitized by Google

There is the method of simple suture, which I consider to be far the best—it is performed either in front or behind the colon; there is the anastomosis by Murphy's button, which I reserve for desperate cases when time is an object, because it can be done in ten minutes; and there is the method by McGraw's elastic ligature, which has no advantage, so far as I can see, over the direct suture.

I think there is often a tendency to over-elaborate every operation, and we have a striking example of this in the method by which colotomy is now done. It used to be a long and tedious operation with a multiplicity of sutures; now it is reduced to its lowest possible terms, and the operation is completed in a few minutes; yet so far as I can see the results are just as good as ever they were. So it is with gastro-enterostomy. I did as little as possible to this man compatible with the end I had in view, which was to unite his stomach and jejunum in such a way that food might readily pass from the stomach into the intestine. I did not expose his colon, I did not cut away the mucous membrane of his stomach, and I did not make large openings. Theoretically, therefore, he ran an additional risk of intestinal obstruction from the jejunum and stomach pressing upon his transverse colon at the point where they were sutured together; the swelling of the mucous membrane at the edges of his wound might have obliterated the aperture; he might have vomited repeatedly by the establishment of "a vicious cycle;" and the hole between the stomach and the intestine might have been too small. None of these things happened, and he stands before you a healthy man, free from pain and vomiting, able to do a day's work again.

Perforated Gastric Ulcer.

You all know how active is the surgical treatment of an acutely perforating gastric ulcer, and how many cases come into the hospital; and those of you who saw me operating upon a case which is now in Kenton Ward will recognise how necessary it is to act promptly. Yet the diagnosis is by no means always easy, and I do not think that any case has come under my care in which some of you have not come to me and said, "Why are you going to operate here? Are you sure that there is a perforation?" I always give you the same answer: I am going to operate because there has been a sudden and violent attack of abdominal pain. The pulse is rapid, and its rate is increasing. The patient looks bad, and his abdomen is more rigid than it ought to be. I care nothing about his temperature, and I do not much want to know whether he has suffered previously from indigestion, or even if he has vomited.

This girl was æt. 20 when she was admitted into the hospital on the evening of November 27th, 1900, suffering from acute abdominal pain. The history obtained with the patient was that she had suffered pain after her food for some months past, but that she had never been sick. She had an attack of severe pain on November 23rd, which compelled her to go to bed, though she got up on the following day feeling better. On November 27th she had a meat lunch, and at 6.30 p.m. she was again seized with acute abdominal pain whilst stooping before the fire. The pain was felt in the epigastrium, and she was sick. She was admitted to the hospital at 11 p.m., when she was found to be anæmic and in great pain. Her abdomen was motionless, but it was neither hard nor distended. The liver dulness was present, and there was epigastric tenderness. The temperature was 98·6° F., the pulse 105, and the respirations 28. I opened the abdomen in the middle line above the umbilicus at 12.30 a.m., six hours after the onset of the symptoms. Some gas escaped immediately, but there was no other evidence of any extravasation of the stomach contents. After a short search a perforation was found on the anterior wall of the stomach towards the pyloric end and near the lesser curvature. There were some flakes of lymph and recent adhesions in the region of the perforation. The stomach was drawn out of the wound, and some blood-stained viscid fluid was then seen to be escaping from the perforation. The hole was closed by two tiers of Lembert's sutures, and after sponging the stomach the abdomen was closed by two layers of sutures. The patient stood the operation well, and on her return to the ward her temperature was 100° F., her pulse 120, and her respirations 44. She received a hypodermic injection of five minims of morphia.

The patient was given nutrient enemata (4½ oz.) every four hours, the rectum being washed out every twenty-four hours. Nothing was given by the mouth for twenty-four hours, but her mouth was frequently sponged out with warm water acidu-

Digitized by Google

lated with lemon juice. After the expiration of the first twenty-four hours a drachm of hot water was given every hour, and after forty-eight hours a drachm of Benger's food. This was gradually increased, and on December 10th the patient was taking solid food and the nutrient enemata were discontinued. The pulse only twice rose above 120, and the temperature varied between 98° and 100° F.

On dressing the wound for the second time on December 11th some thick pus oozed out in two places. A small sinus was found, which was plugged for a few days. The wound was almost healed on January 10th, when a stitch came away; and it was quite healed when she went to the convalescent home at Swanley on January 16th, 1901. Her appetite was then good, and she was free from pain after food. To-day (July 27th, 1903), more than two and a half years after the operation, she tells us that she is well and hardly has an attack of indigestion. The scar in the abdomen is quite firm and is markedly depressed.

It is curious to notice how rapidly the operation of suturing a perforated gastric ulcer has come into favour with us. I find on looking through the statistical tables of the hospital that in 1897 no operation was done; in 1898 one operation was performed, and it ended fatally; in 1899 one man and four women were operated upon, with two recoveries and three deaths; in 1900 four women were operated upon, with two recoveries and two deaths, and this patient is one of those who fortunately escaped. In 1901 ten operations were performed, four on men and six on women; four of the cases died and six recovered. Last year seven women and two men were operated upon for perforated gastric ulcers, three of whom died and six recovered. This year I expect the number will be greater, for I have myself operated upon two men and three women, and four of these, I am glad to say, have recovered.

Cancer of the Tongue.

Many of you will remember this patient. He is a porter æt. 46, who first came to see us in March, saying that he noticed a pimple on his tongue last December. He thought his tongue had been scratched by a sharp tooth which was removed before he came here. The soreness of his tongue increased in spite of full doses of potassium iodide, and it increased so rapidly that I urged him to come into hospital. Mr. G. L. Cheatle, C.B., has lately been pointing out some curious relationships between rodent ulcer and nerve distribution, and I thought this case would be a good one to ascertain whether the division of the nerves going to the tongue had any effect upon the cancerous growth. He consented willingly enough to the operation when I explained that it might be possible to save his tongue by this means, and in April I cut away half an inch of his lingual and half an inch of his hypoglossal nerves on the left side. The wound healed by first intention, and he left the hospital seventeen days later.

The effect of dividing the nerves was to cause atrophy on the left side of the tongue, and coincidently with the atrophy there was a marked decrease in the rate at which the epitheliomatous ulcer was spreading. Before the operation it grew so rapidly that I was eager to get him under constant observation as soon as possible; after the operation the growth seemed to share in the general atrophy and grew slowly, becoming at the same time much more circumscribed. There was no affection of the lymphatic glands below the jaw.

Although the progress of the growth was retarded, it was not arrested, and I feared that if I left it the growth might give rise to secondary deposits. He therefore came into hospital again, and I removed the left half of his tongue, tying the lingual artery on the face of the stump. The operation was performed on June 5th, and the patient left the hospital on June 14th.

A subsequent examination of the tongue showed that the growth was so circumscribed that it would have been the easiest thing in the world to shell it out and leave a small but deep ulcer on the side of the tongue. This specimen shows you that the same thing happens when a cancer of the breast is treated with caustic. The specimen comes from the museum (No. 3181), and the paste was applied by "cancer curers" to a patient who had refused operation. I should like to know whether both in the tongue and the breast the limitation was not due to the nerve action upon the cancer, for we know that arsenic and zinc salts destroy the nerves very effectually, and these are the caustics chiefly used by cancer curers.

Mr. Cheatle was interested in the case, so he attended both operations and carried away the piece of tongue removed. He tells me that a microscopic examination shows the growth to be undoubtedly epitheliomatous, and that the cornifying process is well marked, for there are many

Digitized by Google

epithelial cell nests. The effect of the division of the nerves, then, seems to have been twofold in this case. In the first place it made the cancer grow more slowly and so allowed it to cornify; in the second place it seems to have limited it so completely that if I had waited a little longer I verily believe that the main part of the growth might have sloughed out. He has now a very respectable tip to his tongue, and the tongue is very freely movable, for it is not tied down at all; and if you put your finger at the back you will find that there is no thickening or hardening on the floor of the mouth, and I cannot detect any enlarged lymphatic glands beneath the jaw. However, I believe there is some periostitis in the jaw, and a little scar tissue at the point where the nerves were divided. The man has had all his bad teeth removed from the left side. I think the shrinking of the growth was due to it having taken part in the general atrophy which followed division of the nerves.

Papilloma of the Tongue.

Here is another tongue case. This man is 42 years of age, and three months ago he noticed a swelling on the right side of his tongue. It is not painful and does not appear to get any larger. He has been treated for dyspepsia, but has had no trouble in connection with his teeth. It is on the lower aspect of his tongue, on the right side. The question is, is it a growth, or is it simply inflammatory? It is a pedunculated innocent growth, with processes which show it to be papillary. As it is not malignant you are not surprised to find no enlargement of the glands. You will notice also that the man can put out his tongue and move it freely in all directions. We can soon cure the condition by passing a silk ligature round the pedicle and snipping the growth off with a pair of scissors.

Chronic Gonorrhœal Epididymitis.

This man, æt. 34, has his scrotum swollen. He has been wearing a truss for twelve years. His epididymis is thickened on the left side, but there is no varicocele. On the right side he has a larger swelling, which certainly contains fluid. So he has a hydrocele. Still, it is not an ordinary type of hydrocele, but is probably associated with a long-standing inflammation. He has double and chronic epididymitis, with thickening of the cord. Three years ago, he tells us, he had trouble with his testicle on the right side. I think it is clear also that he has some orchitis. He says he is not losing weight and has no cough. Rectal examination shows that his vesiculæ seminales are not enlarged, so he is not suffering from tuberculous testicle. I judge the case, therefore, to be one of chronic orchitis, with double epididymitis, probably gonorrhœal in origin. The treatment is to tap his hydrocele first, then to give him iodide of potassium and quinine, and afterwards he can have it strapped.

Chronic Osteitis of Tibia.

The next patient is æt. 58, an engineer by occupation. He has been five years under the treatment of Dr. Tooth for hemiplegia. Three months ago he had a small swelling, which commenced of the size of a pea over the left tibia. This gradually became worse, and now it is extremely painful. You will notice that the left leg is larger than the right and that the swelling shades off, but the skin is not red, though the leg feels hot, and it is obviously very tender. He has, of course, periostitis, or, as I prefer to call it, a localised osteomyelitis. The treatment we shall adopt will be to cut down upon the swelling and gouge the underlying bone, the operation being called linear osteotomy. This operation cures the pain by relieving the tension in the bone. I have very little doubt that we shall find thickened periosteum and sclerosed bone, and there might also be an abscess or a small sequestrum of bone; but such sequestra usually lead to the formation of a cloaca and sinus, and in this case there is neither.

Malignant Disease of the Face.

Our next patient is a man 87 years old, who is very well preserved, for he does not look more than sixty. But he comes with a large swelling of the right orbital region and cheek, of three months' duration. At first he thought he had a cold in his eye, but it did not get better. It is increasing in size and discharges a little. His faculties are alert and good, and he does not remember any blow on that side of his face. There is slight proptosis and the veins are enlarged. The swelling is diffuse and œdematous. There is, too, considerable chemosis. I think there is some slight facial paralysis, but he can blow down the right nostril. The swelling is probably due to a sarcoma, especially when we consider his age. If the swelling had been inflammatory or tuberculous there would have been a considerable quantity of pus there by now, as it is of three months' duration. I cannot feel any enlarged glands. It is desirable to find out whether it is growing from the antrum, and for that purpose we must use transillumination. If it is not antral—and I do not for a moment suppose that it is,—the sarcoma is periosteal, and springs from the superior maxilla. He says it causes him so much distress at night that he cannot sleep. Our object should be to promote his euthanasia, for no operation will help him, and to this end I order him Pil. Saponis Co., gr. x, to be taken every night at bed-time, and I tell his daughter to take good care of him as she will not have a father much longer.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 574. WEDNESDAY, OCTOBER 28, 1903. Vol. XXIII. No. 2.

CONTENTS.

PAGE

*Some Clinical Considerations concerning certain of the Rarer Forms of Enlargement of the Heart. By W. HALE WHITE, M.D., F.R.C.P. ... 17

*A Lecture on Epilepsy and its Treatment. By JAMES TAYLOR, M.D., F.R.C.P. 26

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½ *Bartholomew Close, London, E.C.*

SOME CLINICAL CONSIDERATIONS CONCERNING CERTAIN OF THE RARER FORMS OF ENLARGEMENT OF THE HEART.*

By W. HALE WHITE, M.D., F.R.C.P.,
Physician to Guy's Hospital.

GENTLEMEN,—In the first place I must thank you for the honour you have done me in asking me to give you an address to-night. I assure you I feel that an invitation to such an old society as this is a very great distinction.

In the next place I must apologise for my choice of subject, for the difficulties connected with it are so many that it is impossible for me to treat it satisfactorily; but my excuse must be its importance. This is so great that probably good only can follow our thinking about it. With regard to my selection of the word enlargement rather than hypertrophy or dilatation, I may adopt to my own needs the words that Dr. G. H. Barlow† wrote in 1847. He says, "Before proceeding further I would remark that I use the term enlargement, not only because I always prefer an English word where one can be found to suit my purpose, but also because it may, without impropriety, be made to include both hypertrophy and dilatation—lesions which, though essentially different, are so commonly united that it is often difficult to treat of them separately."

Considerable experience of examining medical students for their final examination in medicine has revealed to me that most are taught that they can accurately determine by percussion whether a heart is enlarged. The first text-book I looked into after writing this sentence says, "The area of cardiac dulness is increased in all conditions where

* Delivered before the Hunterian Society, October 14th 1903.

† 'Guy's Hospital Reports,' vol. v, ser. 2, p. 173.

Digitized by Google

the organ is enlarged." I think this is not so. In fat women with big breasts I would submit that it is often impossible to infer by percussion whether the heart is enlarged; then, again, emphysema may prevent our detection of cardiac enlargement. Let me quote one of our greatest clinical observers. Fagge* says, "Now it must not be supposed that percussion affords an infallible indication of enlargement of the heart, even when the lungs are healthy, being neither emphysematous nor retracted. On the contrary, very great hypertrophy of the left ventricle may be present (as, for example, in cases of Bright's disease) without there being any increase in the area of dulness. The organ seems to bury itself within the hollow of the left lung, so as not to come more widely into contact with the chest wall than in normal circumstances." My own experience certainly agrees with this. Not only does percussion often fail to detect cardiac enlargement, but unless used with care it may lead us to falsely infer the presence of cardiac enlargement, for the lungs may be retracted from the heart, or pulmonary consolidation in the neighbourhood of the heart may give a false impression of cardiac dulness. I believe that in many instances, unless enlargement of the heart is detected by other means than percussion, this will not help us to a certain conclusion; and this applies to both superficial and deep percussion.

Naturally, the first section of our subject will be the influence of diseases of the pericardium on cardiac enlargement. This structure is too often regarded solely as a serous sac which by its smooth surfaces allows cardiac movements to take place with little friction. But this must be an unimportant function, for it is allowed by all who have a considerable experience of morbid anatomy that the two layers of the pericardium may be adherent in those who show no signs of impaired circulation. Wilks and Moxon† say, "The heart is found universally adherent to its serous covering by means of a most delicate areolar substance, and no evil appears to result from it. . . . . Our own opinion is that simple pericardial adhesion produces no appreciable untoward consequences." Sequira‡ says, "It is quite a common thing to meet with a universally adherent pericardium in the post-mortem room in a case in which the heart was supposed to be healthy." It will always be found that, if symptoms indicating difficulty of circulation occur in association with adherent pericardium, either the valves of the heart are diseased, or that organ is enlarged, or its muscular substance is diseased, or the pericardial adhesions have strangled the large veins as they enter the heart, or lastly, the adherent pericardium is associated with adhesions of other serous membranes.

Thus we see that the pericardium does not owe its importance to its being a serous membrane. It is of value as a support to the heart, and to prevent its over-distension. This has been made quite clear by the work of Leonard Hill and Barnard, for if the pericardium be removed it is possible to distend the heart more than before. The pericardium prevents the over-distension of the heart that would otherwise occur during severe muscular effort. The great importance of this in thinking of the treatment of pericarditis we shall see later.

When pericarditis occurs the pericardium becomes softened and sodden, and hence it no longer forms so strong a support to the heart. Further, there can be no doubt that pericarditis is often associated with myocarditis due to direct spread from the visceral layer of the inflamed pericardium; this can commonly be seen in the post-mortem room by the naked eye, especially if the patient has lived some time after the onset of pericarditis; then a layer of fatty cardiac muscle, often one sixteenth of an inch thick, is seen under the inflamed pericardium.

Probably, too, in rheumatic cases, myocarditis occurs sometimes quite apart from pericarditis; inded, if we are to rightly apprehend the treatment of rheumatic heart disease, we must remember that rheumatism leads to endocarditis, myocarditis, and pericarditis, although it is not by any means necessary for all three to be present in the same case. Now as both the wall of the heart and the pericardium are softened and weakened by pericarditis, it is little wonder that the heart—no longer able to withstand the intra-cardiac pressure—dilates. Dilation must, unless it rapidly passes away, lead to hypertrophy; for, as Leonard Hill* points out, "the total fluid tension to which the

* 'Principles and Practice of Medicine,' vol. ii, p. 24, 1st edit.

† 'Pathological Anatomy.'

‡ 'Trans. Roy. Med. and Chirurg. Soc.,' vol. lxxxii, p. 408.

* Allchin's 'Manual of Medicine,' vol. iv, p. 299.

Digitized by Google

wall of the heart is submitted rapidly increases as the radii of curvature become greater. A heart when distended must therefore put forth far greater energy in order to raise the contained blood to the pressure of that in the aorta. Hence a dilated heart fails to empty itself, and hypertrophies." Thus we see that if the heart begins to dilate in a case of acute pericarditis, it will soon also hypertrophy; and most writers are agreed that, quite apart from any concurrent endocarditis, dilatation and hypertrophy of the heart are frequent results of pericarditis. For example, Dr. Goodhart* has recorded a case which seems to show that a heart grew so that it weighed nineteen ounces at death as a result of pericarditis. But there is worse to come in many instances. Probably in every case, and certainly in every severe case of pericarditis, adhesions form between the visceral and parietal layer. If these adhesions are of the tough, firm variety, and they take place when the heart is dilated, they will hold it out, and it can then never, or only with great difficulty, recover its normal size, and the patient becomes a permanent invalid with a form of heart disease that no treatment can touch; and the matter is still worse if, as is often the case, pleuro-pericarditis has led to abnormal adhesions on the outside of the pericardium. We should never plume ourselves that our patient has recovered from pericarditis just because the rub has disappeared and the temperature has fallen. Our treatment has not been successful unless, although adhesions have taken place, the heart is not dilated. With the present means at our disposal we must often fail to attain this, especially when endocarditis is present; but it is the goal to strive for, and when we do not reach it we have failed as a surgeon fails when, after a fracture, the patient is a cripple because union has taken place in a bad position.

Let us now consider how to avoid these difficulties and how best to secure that adhesion shall take place when the heart is not dilated. As dilatation occurs because, owing to softening of the pericardium, the heart has lost its natural support and its own muscle is weakened, we must keep the intra-cardiac pressure as low as possible; exertion raises the pressure considerably, hence the patient must be kept absolutely quiet on his back. He should never sit up, and on the rare occasions when his back is examined he should be rolled over. He must never get out of bed, not even when the bowels are moved. Defæcation and vomiting both raise the blood-pressure, and hence tend to dilate the heart. Therefore it is extremely bad treatment to allow the bowels to become constipated or to allow the patient to strain at stool. The bowels should be open at least once a day, the motions should be soft; and all this can be attained by giving cascara, senna, or castor oil. Great care must be taken to prevent vomiting, and hence anything—such as digitalis—which may cause it should be given by the rectum if it induces nausea. Food should be given in small quantities at frequent intervals; and it is particularly necessary to avoid flatulence, for a distended stomach pushes upon the heart. Alcohol should not as a rule be given, as it raises the blood-pressure, and inasmuch as this is lowered during sleep the patient should be kept gently under morphia. Sleep has a further advantage, for during it the rate of the pulse is slowed, and hence both the inflamed pericardium and heart are rested. Opinions vary as to the advisability of giving digitalis; for my own part I feel sure I have seen benefit follow in severe cases. The great advantage of it is that it contracts up the dilated heart. We have just seen at what a rapid ratio the work of the heart increases as its cavity dilates; hence the advantage of contracting it by every means in our power. Further, the whole object of treatment is to get adhesions to form on the organ when it is contracted rather than when it is dilated; and likewise it is important to have a long diastole, for the adhesions can obviously become better organised during the rest of diastole than during systole; also there is often some myocarditis present with the pericarditis, and digitalis, by prolonging the diastole, increases the time during which reparative metabolic processes can take place in the heart; and lastly, inasmuch as during systole blood is expelled from the coronary arteries by contraction of the heart, a drug which diminishes the frequency of systole and prolongs diastole probably facilitates the blood-flow in the coronary arteries, and so helps the repair of the diseased heart. Cases I have seen have taught me that these advantages of digitalis quite outweigh the disadvantages of raised arterial pressure, and further, if this is marked, strophanthus may be given instead of digitalis.

* 'Path. Trans.,' vol. xx.

Digitized by Google

The next point I wish to urge is the importance of prolonging the treatment. Let us always have before us the permanent invalidism which will follow if the heart dilates and remains dilated, and then we shall not be in a hurry to get the patient up, and thereby raise his blood-pressure too soon. As the heart is constantly contracting, it seems only reasonable to believe that recovery from myocarditis will be slow, and the sodden pericardium must have time to again become firm. Probably very few sufferers from pericarditis should be allowed to sit up in less than six weeks after the temperature is normal; in others a longer rest is necessary. At first propping up in bed is all that can be allowed, and only a few mimutes at a time of that; then, as days go on and no harm follows, the patient may sit in a chair, and next gradually get on to his feet. Much care must subsequently be shown in regulating the amount of muscular work he may gradually do, but throughout we must hasten slowly. I need not stay to emphasise how much greater is the danger from pericarditis if valvular disease is also present, nor to remind you that pericarditis is much more serious in children than in adults. Probably more than one cause is at the bottom of this. In the first place, the pericardium is less strong than in adults, and therefore when inflamed cannot so easily withstand cardiac pressure; hence it rapidly dilates, and consequently hypertrophies. The heart of a child afflicted with pericarditis may double its weight in three weeks. Secondly, even if the pericarditis quickly subsides, it is unlikely that the pericardium, having been once acutely inflamed, will be able to adapt itself as readily as it should to the physiological growth of the heart, and hence, as Sequira has shown, if a child has had pericarditis the heart breaks down at the period of youth, when growth is most rapid.

Leaving now the pericardium, and passing on to the heart and vessels, I may at once mention that I do not propose to say anything about the enlargement associated with valvular disease, with chronic nephritis, or general arterio-sclerosis, for it is so well known to you and the subject is so large; but I cannot resist pointing out that many patients with enlargement due to chronic interstitial nephritis do not give themselves a fair chance, for the condition is so slow in its development that they forget to obey their doctor's orders, directed to keeping down the blood-pressure,—for example, they over-exert themselves, they eat and drink too much, and do not keep their bowels comfortably open.

Omitting all these common causes of enlargement, we next have to consider the effect of toxins on the heart; and we are all familiar with the fact that in many specific fevers, notably scarlet fever, typhoid fever, diphtheria, pneumonia, and influenza, the heart dilates, its action becomes feeble, rapid, and irregular, and the patient frequently dies from cardiac failure and an actual myocarditis. In this condition the physician can often by judicious treatment save the patient's life. He must be kept still in bed, he must not be sat up on any consideration, and if he be desperately ill and passing everything under him, it may be well to leave him for a few hours with his drawsheet unchanged rather than disturb him. Strychnine is of the very greatest value; it should in a very severe case be given even until the patient shows slight signs of poisoning by it, and it has the great advantage that it can be administered subcutaneously. Digitalis, too, is very helpful, but it has disadvantages not possessed by strychnine, for it is not so easily given subcutaneously, and it may make the patient sick. Caffeine or strong ammonia is, I think, very useful, and a good prescription consists of 5 minims of liquor strychninæ, 15 or 20 minims of tincture of digitalis, with 5 grains of caffeine and 2½ grains of salicylate of sodium to ensure the solution of the caffeine to an ounce of water. Another useful mixture consists of 4 drachms of the infusion of digitalis with 4 minims of strong solution of ammonia to the fluid ounce of water. Either of these may be given every four hours with, if necessary, some strychnine subcutaneously. These drugs are, I think, better than alcohol, for it is impossible to tell beforehand whether alcohol will suit a patient, and a few hours are lost while we are watching to see whether or not he is improved by alcohol. Sleep is very important, and hence the value of a subcutaneous injection of morphia.

But inasmuch as this condition of heart is so well known, and also the consideration of it falls more under that of specific fevers, we may now consider the action of other poisons; and I should like to direct your attention to a form of heart disease which has received very little notice in this country—namely, that due to over-indulgence in alcoholic drinks. Many English writers either

Digitized by Google

ignore it altogether or merely have a line to the effect that alcohol, tea, coffee, tobacco, digitalis, uric acid, and other poisons may cause hypertrophy and dilatation of the heart. They often do not draw attention to the seriousness of the alcoholic heart, and they hardly ever adduce any evidence that these poisons have any influence in the production of heart disease. But German physicians are well aware of alcoholic heart disease, and German writers describe it fully. Osler refers to their description, and makes the interesting observation that Strümpell at his Erlangen clinic told him the condition was very common in that town among those who worked in breweries. Perhaps I shall best introduce the subject to you by reading notes of four cases that have been under my care.

Case 1.—Charles H—, æt. 44, was admitted into Stephen Ward under Dr. Hale White at 9 p.m. on June 25th, 1896. He was collapsed, and it was learnt that he had been much addicted to alcohol. The heart-sounds could not be heard, and the pulse was imperceptible. His breathing was stertorous. His extremities were cold and his legs œdematous. He died at 3.30 a.m. on June 26th. The post-mortem examination was made the same day. The heart weighed twenty-two ounces. The muscle was pale, and there was a considerable deposit of fat on the right ventricle. The coronary arteries appeared healthy. The right ventricle was thickened, it was pale, and streaked with fat. The tricuspid orifice measured $5\frac{1}{2}$ inches, the pulmonary $3\frac{1}{4}$ inches. The wall of the left ventricle was hypertophied over three quarters of an inch thick. Muscle pale and fatty. Apex of musculi papillares pale, light yellow, and below this deep purple from streaks of hæmorrhage. The mitral orifice measured $4\frac{1}{2}$ inches; there was no endocarditis. The heart showed, when examined histologically, fatty and pigmentary degeneration of muscle-fibres. The arteries were not atheromatous. The kidneys were healthy. The liver was very fatty; it floated on water.

Case 2.—Thomas M—, æt. 50, was admitted into Guy's Hospital under Dr. Hale White on November 12th, 1896. He came in on account of swelling of the legs. He was fairly temperate till two years ago, but since then he has been a very heavy drinker. He first began to feel ill six or seven weeks ago. Three weeks ago his legs began to swell. On admission he had the aspect of a drinker; his hands were tremulous and he was very short of breath. Temperature 100°; respirations 34; pulse about 120, very irregular, feeble, and small. The edge of the liver could be felt three inches below the costal margin. No ascites could be detected, and the spleen could not be felt. There was a trace of albumen, and the specific gravity of the urine was 1024. There was some viscid expectoration, and there were some fine râles at both bases. The pulse was so irregular that accurate counting was impossible. The heart's impulse was feeble, but no murmurs could be heard. The cardiac dulness was increased to correspond with the position of the impulse. There was œdema of the feet. The reflexes were all normal. The diagnosis made was dilatation and hypertrophy of the heart from alcoholism; swelling of legs and slight albuminuria secondary to the cardiac trouble; the cardiac dilatation gradually diminished (as shown by diminishing cardiac dulness and shifting in of the impulse). The albuminuria disappeared in a day or two. On November 19th he became so noisy and restless that he had to be removed to the strong room; he soon showed well-marked signs of delirium tremens, his temperature rose to 103°, and he died on November 23rd. During the whole of his stay in the hospital his pulse remained rapid, irregular, and weak. At the autopsy it was found that the arteries of the brain and the coronary and pulmonary arteries were atheromatous; otherwise the arteries of the body were not noticeably diseased. The lungs were tough and red, and showed general evidence of backward pressure. The liver was nutmeg. The kidneys were large but healthy. The heart weighed twenty ounces. The left ventricle was much dilated and hypertrophied. The valves were all healthy. The tricuspid orifice measured 6 inches, the mitral $4\frac{3}{4}$ inches.

Case 3.—Charles H—, æt. 34, was admitted into Philip Ward under Dr. Hale White on May 19th, 1903, for œdema of the legs and shortness of breath. Clinical clerks, Messrs. R. A. Chisholm and G. H. MacAlister. His family and personal history is unimportant, except that formerly he used to be rather a heavy drinker, but for the last nine months he says he has only taken a little brandy. He has had no rheumatic manifestation, but he has had gonorrhœa and syphilis. Nine months ago he had to give up his work (that of an

Digitized by Google

engineer) on account of shortness of breath, which had been coming on gradually. At the same time he noticed that his eyes and face were swollen. Since then his trouble has increased, and his shortness of breath is now very marked, and he becomes cyanosed on exertion. He has once coughed up a little blood and is troubled with a cough.

On admission he is well nourished. His respirations are rapid, his lips cyanosed, and he has œdema of the ankles and thighs. The cardiac impulse can be both seen and felt an inch outside the nipple line and in the sixth space. No murmurs can be heard even when the patient walks up and down the ward, but he cannot do this for long, as it brings on severe dyspnœa. There is often a triple beat, and the heart's action is very rapid and often irregular. When he first came in, the force of the pulse was fairly good and the tension normal, but gradually the pulse became feebler and feebler till his death on June 15th. His liver reached to within an inch of the umbilicus; it was tender and uniformly enlarged. His urine was considerably diminished in quantity; the specific gravity was usually above the normal; albumen was always present in fair amount. He suffered from diarrhœa. Four days before he died he began to cough up much blood, and this continued till his death, which occurred from cardiac failure. He had during most of the time a few râles in his lungs. He was thought during life to be suffering from an alcoholic heart with secondary enlargement of the liver and congestion of the kidneys.

At the autopsy the heart was found to be uniformly enlarged; it weighed twenty ounces. The valves were normal. The liver was nutmeg. The kidneys were the hard, indurated kidneys of heart disease. There was a large pulmonary apoplexy. The coronary arteries were not atheromatous. Histological examination of the cardiac muscle showed nothing abnormal.

Case 4.—A publican æt. 60, seen September, 1903, with Dr. F. H. Bence. He had drunk hard, and when we saw him he was dying. He was sitting up very short of breath, with a feeble pulse of 150. The cardiac sounds were faint; some emphysema made the estimation of cardiac dulness uncertain. Save for the emphysema the lungs were healthy, there was no albumen in the urine, the arteries were not thick, the liver was somewhat enlarged, there was very little œdema of the feet, and no sign of peripheral neuritis. No post-mortem examination could be obtained, but the man's condition strongly suggested an alcoholic heart.

Now you will notice that all these patients were hard drinkers; the first three were men who were admitted into the hospital for disease of the heart. In none could any murmurs be heard; but in those that lived long enough for a thorough clinical examination, much cardiac dilatation and hypertrophy was shown by the position of impulse and the extension of cardiac dulness. There was nothing to indicate that adherent pericardium was the cause of the trouble; the failure to obtain a history of rheumatic fever, and the absence of murmurs, showed we were not dealing with valvular disease; the absence of thickening of the arteries and the character of the pulse made it unlikely that the patients were suffering from general arterio-sclerosis; nor did the urine have the characters which are usually associated with chronic interstitial nephritis; and the examination of the eyes showed no albuminuric retinitis. None of these conditions were found at the autopsy, so we are right in assuming that the diagnosis made during life of "alcohol heart" was correct. You will notice what we found post mortem shows that an alcoholic heart is one in which there is considerable hypertrophy and dilatation (the organ weighed twenty-two ounces in the first case and twenty ounces in the second and third) without any obvious cause—such as valvular disease, arterial disease, Bright's disease, or pericardial disease—being found.

From these patients we can deduce the symptoms. The most striking are that the patient is very short of breath and has a rapid feeble pulse. The dyspnœa is frequently very extreme; the patient finds it impossible to lie down at night, and if he drops asleep he wakes up with a start panting for breath. The pulse frequently beats 150 to the minute; it is feeble and small and usually regular, although at times markedly irregular. Very slight exertion enormously increases its rate. On examining the patient it is found that the heart's dulness is increased; the impulse is diffuse, feeble, and displaced outwards; the cardiac sounds are very weak, but as a rule no murmur is present; the second sound may be reduplicated.

Digitized by Google

The patients complain much of a sense of oppression or actual pain in the chest, of palpitation and of fulness in the epigastrium. It is surprising how ill they may be before any signs of backward pressure show themselves, and they may die before they are seen. When they do appear they are such as are commonly met with in heart disease,—for example, œdema of the feet, an enlarged nutmeg liver, lividity, hæmoptysis, bronchitis, œdema of the lungs, scanty high-coloured urine, with a little albumen; and there is much dilatation, and a murmur indicating mitral regurgitation may be heard.

The post-mortem appearances of an alcoholic heart are striking, for there is considerable hypertrophy of both sides. In our first case the organ weighed twenty-two ounces, in the second twenty, and in the third twenty. I have read of cases in which the heart weighed a pound and a half. By the time death takes place there is dilatation as well as hypertrophy. Sometimes there is associated alcoholic fatty degeneration, as shown by yellow striation; this was very evident in our first case. The majority of authors believe that no important histological changes can be found, and that certainly was so in our first and third cases, which were examined histologically; but some writers attach much value to the fact that the muscle-fibres are widened, the nuclei are increased in size, and the muscle-fibres show fragmentation; but the balance of evidence is that these changes are either post mortem or are produced during the agony, and they are undoubtedly met with in other diseases besides alcoholic heart disease. A few authors have described patches of myocarditis, but they are so rarely present that they must owe their existence to some other cause than the alcoholic drinks.

Having finished this short sketch of the condition, let us inquire a little more fully about it. The older authors described an idiopathic cardiac hypertrophy; then, when it was discovered how very important valvular disease and obvious increased peripheral resistance and pericarditis were as causes of hypertrophy and dilatation, there followed a period when the tendency was to assign all cardiac hypertrophy and dilatation to these. Next Myers, De Costa, Allbutt, and Fraentzel pointed out the effect of strain; and then in 1877 Münzinger called attention to the frequency of dilatation and hypertrophy apart from any of the above causes among the inhabitants of Tubingen, and the phrase "Tubingen heart" came into use. It was soon shown by Buhl and Bollinger that the same kind of heart occurred at Munich, and we had the phrase, "The Munich heart." But, unfortunately for the human race, it was quickly found that this disease was not limited to Tubingen and Munich. I have already mentioned that it occurs at Erlangen; and, indeed, it is met with in many places, especially in Germany, where the inhabitants consume excessive amounts of beer. Occasionally as much as twenty litres, or about thirty-five pints of beer, are drunk daily by a single individual. Bauer* and Bollinger, in their monograph, give many cases, and sometimes the heart weighed as much as 790 grammes, which is roughly a pound and a half. Bollinger found 244 cases of fatal alcoholic hypertrophy and dilatation of the heart among 5720 post-mortems,—that is, in about 4 or 5 per cent., the great majority of the cases being men. It is generally believed that these hearts show no important histological changes, but lately observers have attributed much importance to patches of interstitial inflammation, which, as already mentioned, were occasionally found scattered throughout the heart. This part of the subject is very fully described by Albrecht,† who believes that alcohol is the cause of the inflammatory changes; but he quotes the opposite opinion of Krehl, who, taking his ground on the fact that the administration of absolute alcohol will not lead to these changes, suggests that the alcohol lowers the resistance, so that the heart easily becomes the prey to bacterial invasion, and hence the inflammatory changes; and inasmuch as Krehl gives myocarditis as one of the conditions which an alcoholic heart has to be distinguished from, we see it is by no means universally allowed that alcohol causes a myocarditis. Another possibility which ought, I think, to be discussed, is that the cardiac trouble is dependent upon alcoholic neuritis of the vagus. This undoubtedly occurs—indeed, it is one of the causes of death in alcoholic neuritis. The pulse becomes irregular, rapid, and weak, the patient dying from cardiac failure; but probably the sufferers from an alcoholic heart do

* "Ueber Idiopathische Herzvergrösserung," von Pettenkofer's 'Festschrift. München,' 1893.

† 'Der Herzmuskel und seine Bedeutung für Physiologie, Pathologie, und Klinik des Herzens,' Berlin, 1903.

Digitized by Google

not owe their condition to neuritis of the vagus, for most of them are males, whilst most of the sufferers from alcoholic neuritis are females; and also it is quite the exception to find that those who have an alcoholic heart also suffer from peripheral neuritis.

Tresilian,* in 1898, briefly recorded five cases, in none of which was there an autopsy, but in which there was a rapid pulse, signs of cardiac dilatation, and evidence of cardiac failure. His first and fifth cases were undoubtedly examples of neuritis of the vagus, for the patients were women suffering from peripheral neuritis. The nature of the other cases is doubtful; probably they were examples of the alcoholic heart described by the Germans, but Tresilian makes no reference to this condition. I have read many cases of the alcoholic beer heart described by the Germans, and inasmuch as other signs of peripheral neuritis are not recorded I think the evidence is all against the supposition that this condition is due to alcoholic vagal neuritis, with which we are perfectly familiar as a sequel of ordinary alcoholic peripheral neuritis.

Another question that arises is whether the increased quantity of fluid which must pass through the vessels of those who drink such large quantities will cause cardiac hypertrophy; but the body so quickly gets rid of an excess of fluid in the vessels that we have no certain proof that the total amount in the vessels at any time is much above normal; and then, again, the work of the heart is the resultant of the quantity of blood and the rate of flow, and if the quantity is increased it may be that the rate of flow is diminished, for a rapid pulse does not necessarily mean a rapid flow. Another reason for doubting whether a large amount of fluid in the vessels—a hydræmic plethora—will lead to dilatation and hypertrophy of the heart is the condition of that organ in diabetes. Many diabetics drink largely and have polyuria, and yet the heart is usually small in this disease, sometimes very small. It is true that diabetes is a wasting disease, but at any rate we can say that the large amount of fluid passing through the vessels does not lead to sufficient cardiac hypertrophy to prevent the wasting attributable to the diabetes. Bearing, too, on this question is the fact that those who drink excessively of water—for example, the stokers of vessels in the tropics—do not suffer, as far as we know, from cardiac dilatation and hypertrophy.

In passing, I may mention that it has been suggested that the carbohydrate in the beer is the cause of the hypertrophy, but if this were so we should expect vegetarians to have enormous hearts, but I have never heard that they do. The disease of the heart has been laid at the door of the salts in the beer, but of this there is no evidence. Indeed, we have no clue as to the constituent of beer which is the cause of the trouble. We are in the same difficulty here as in other diseases which follow excessive drinking of alcoholic drinks—namely, that absolute alcohol administered to animals will not produce, for example, cirrhosis of the liver; and the kind of disease resulting from drinking differs in different places. Thus cirrhosis of the liver is common in London but rare in Scotland, while an alcoholic heart is common in Munich but not in Great Britain. Until the various evil results of over-drinking can be reproduced by absolute alcohol it will be better to keep an open mind on the question as to which constituent of alcoholic drinks is the exciting cause. Before leaving this branch of our subject I ought to mention that Bauer is inclined to attribute the hypertrophy partly to increased peripheral resistance caused by the alcohol, but certainly in some cases the pulse is of low pressure.

The diagnosis need not detain us long. The history of drinking, the absence of a murmur till late in the case, will serve to distinguish alcoholic heart disease from mitral disease, and there can hardly be any difficulty with regard to aortic disease. In some, chronic interstitial nephritis will much resemble alcoholic heart disease, but usually there are many differences. It is said that the diagnosis between myocarditis and alcoholic heart disease has sometimes to be made, but acute myocarditis always follows a specific fever, and chronic myocarditis or fibroid heart is rare, and does not cause a rapid pulse or great dyspnœa; indeed, some writers have asserted that the pulse is slow in cases of fibroid heart. Fatty degeneration of the heart, if general, is usually associated with general anæmia, wasting diseases, as phthisis or cancer, or with over-eating, and in any of these the history will put us on the right track; but it may be due to chronic alcoholism, and then may be difficult to distinguish from the alcoholic heart disease here described. But the fatty heart is not hypertrophied to the same extent; the sufferers are liable to seizures of sudden cardiac failure, and the pulse is not so persistently rapid as in the alcoholic heart. It is said that diseases of the coronary artery may give rise to difficulty, but the history and the increased size of the heart will probably be of help in diagnosis. I have not gone into the question of diagnosis more fully, because I always think this is better discussed at the bedside than

* 'Edinburgh Medical Journal,' vol. i, 1898, p. 616.

Digitized by Google

on paper, and partly because I hope to go into the diagnosis from other conditions more fully elsewhere.

The other poisons affecting the heart are tobacco, tea, and coffee when taken to excess; but inasmuch as they do not enlarge the heart they hardly come into consideration here.

The last condition to which I have time to direct your attention is fibroid disease of the heart. This hardly as a rule receives the notice that it deserves, for it is important because it is a cause of sudden death. A man who appears perfectly well may drop down dead, and at the autopsy the only sign of disease anywhere may be fibroid disease of the heart, which often cannot be seen either on the external or internal surface of the heart, and is only discovered on cutting up the organ, especially the interventricular septum. Dr. Fagge's classical paper on the subject appears in the Pathological Society's 'Transactions' for the year 1874. He particularly points out the liability to sudden death; and I recorded in the Pathological Society's 'Transactions' for 1884 the cases of this disease which had died suddenly and had occurred at Guy's Hospital in the intervening ten years. There were nine of them. Four of these nine were brought into the hospital dead, two died in the out-patient room, and of the three who were in the hospital one died suddenly while up and about the ward. Another, who was not thought to be dangerously ill, and had only been in the hospital a day, was suddenly seized with dyspnœa and died in a few minutes; and with regard to the ninth case the patient died unexpectedly. Thus we see that fibroid disease of the heart is a cause not only of sudden, but also of unexpected death. Others have recorded in the Pathological Society's 'Transactions' cases illustrating these points. As death is so often sudden and unexpected we are naturally very ignorant of any definite symptoms that would enable us to diagnose the condition. At the post-mortem examination the alteration is always easily seen, provided that the heart is sliced up; for, as already mentioned, the change is often not visible externally, and is usually best marked in the septum between the ventricles, but it may occur anywhere in the wall of the ventricles. I have never seen it in the wall of the auricles, and it is rarely seen in the right ventricle. The affected part is usually white, hard, and glistening, but in some cases it is softer and somewhat yellow. Under the microscope abundant fibroid tissue is seen, and if this has existed for some time the wall bulges and a cardiac aneurysm results. Often the doctor is not called upon to make a diagnosis, for the patient is not seen until after death; but the heart may fail and backward symptoms ensue, when the case comes to resemble one of mitral regurgitation; or if, owing to congestion of the kidneys, albumen appears in the water, then a patient with fibroid disease of the heart may be thought to have granular kidneys.

It is not to be expected that fibroid tissue would support the intra-cardiac pressure so well as muscle, and therefore we are prepared to learn that the organ may be dilated and the fibrous part of its wall thinned; but it may also be hypertrophied without any dilatation. For example, a man was brought into the hospital dead; absolutely all that was found abnormal at the post-mortem examination was that the heart weighed fifteen ounces, with thick walls, and the ventricular septum was so infiltrated with fibrous tissue that its wall was an inch thick. The left ventricle often becomes very thick and rounded, so that it measures even more in its antero-posterior than in its transverse diameter; but on the other hand there may be no hypertrophy, and the heart may be quite normal in shape It appears that just as the heart will hypertrophy when any difficulty is placed in the way of its doing its work, so when part of it becomes fibrous the rest of the muscle hypertrophies, and the amount of hypertrophy will depend upon the degree to which the fibrous material impairs the working of the heart; and further, if the patient lives long enough for the fibrous material to give and dilate, then the muscle will hypertrophy just as it does whenever the heart dilates. It is easy to see that the weight of the heart will depend upon the amount of hypertrophy and its ratio to the fibrous tissue, and hence we sometimes find the heart about or not much in excess of the normal weight—as, for example, in the case of an elderly, badly nourished man on whom I made a post-mortem, and whose heart weighed thirteen ounces; except for fibroid testes and other signs of old syphilis, the only thing abnormal at the post-mortem was fibroid change in the interventricular septum. His coronary arteries were healthy. A certain number of these fibroid hearts owe their origin to syphilis quite apart from syphilitic disease of the coronary arteries, and in some of the most marked cases the disease is apparently due to old pericarditis; but these form quite a small number, and we are forced to conclude that there is a fibroid disease of the heart which may lead to its dilatation and hypertrophy quite apart from pericarditis, and that syphilis will only explain a certain portion, perhaps a quarter or a third of these.

I must, in conclusion, apologise for the extremely fragmentary nature of this paper; but I thought, and I hope I have been right, that it would be of more interest if I dealt with some rare rather than common causes of enlargement of the heart, and therefore I have left out obvious causes, such as valvular disease, renal disease, arterio-sclerosis, strain, and I have not dealt with cases in which enlargement of the right side was due to pulmonary obstruction.

Digitized by Google

A LECTURE

ON

EPILEPSY AND ITS TREATMENT.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, May 12th, 1903.

By JAMES TAYLOR, M.D., F.R.C.P.,

Physician to the Hospital, to the North-Eastern Hospital for Children, and to Moorfields Eye Hospital.

GENTLEMEN,—In what I have to say to-day I shall not attempt to give a systematic lecture on epilepsy, because it is still so much one of the indefinite entities that it would be hopeless to attempt to do anything of that kind well. I shall content myself simply with talking about the various points in the symptomatology of epilepsy and offering you a few hints, so far as I have been able to glean them from my experience, with reference to the treatment of the disorder. One will naturally dwell, to a certain extent, on the pathology, and will refer also to the prognosis of the malady. I shall not try to define epilepsy, because we all know fairly well what we mean when we talk of it. There are the severe forms of epilepsy, known as *grand mal*, and also slighter forms of it, *petit mal*. And there are various other conditions which ought to be referred to in considering epilepsy, because of the extremely close similarity which the convulsions have in those conditions to those of epilepsy. In the first place we have to deal with convulsions occurring in early life. We know that in the newly born there are sometimes convulsions, perhaps a series of severe convulsions, which pass off, leaving the child seemingly little if any the worse for the occurrence of them. The cause of them is doubtful, but they occur chiefly in children who are born asphyxiated, in the birth of whom there has perhaps been a long and difficult labour. In some of those cases—and I shall scarcely do more than refer to them—there is a definite lesion on the surface of the brain. But the convulsions in early life and in infancy to which I wish chiefly to direct your attention here, are those which occur apart from any definite organic change in the brain, and which pass away and leave practically no trace behind them. Then, besides the convulsions in very early infancy and in the newly born, there are convulsions which occur during the first few months of life. These are associated with rickets, rickets being that form of malnutrition which manifests itself most obviously in the enlargement at the ends of the long bones. What the cause of the convulsions in rickets is we are not sure, but the probability is that they depend upon some instability in the cortical cells, brought about by some form of disproportion between the development of the nervous elements and that of the connective-tissue elements, similar to what occurs in the bones. At all events we know that in rachitic children we have convulsions in every respect closely resembling those which we meet with in later life in what we call epilepsy. It is very important to recognise this, because there is good reason to believe that the convulsions of rickets are sometimes the precursor of true epilepsy occurring in later life; and in all cases of epilepsy it is important to inquire into the previous history of the epileptic patient, more especially with reference to the occurrence of convulsions in early life associated with rickety deformities. Besides rickets in early life and the difficulties attending the entrance of the child into the world, there is a disease unfortunately common in early life—namely, tubercular meningitis,—and in that also we have convulsions. Sometimes the convulsions are general; at other times they are local,—that is to say, of the nature of the convulsions which we shall presently refer to as Jacksonian epilepsy. And it is well to keep in mind those various forms of disease in which convulsions occur, because when one is first called to see a child suffering from a convulsive seizure one has to consider the different forms of disease in which such convulsions occur, in order to be able to determine definitely what the nature of the convulsion in the particular case is. Then in uræmia, due of course to kidney disease, we have convulsions occurring in every respect similar to those which take place in epilepsy. And a case of uræmic convulsions when first seen is difficult, if not quite impossible, to distinguish from a case of *status epilepticus*, because the convulsions are of a similar character to those which we shall presently refer to, and they occur one after another, just as they do so often in that state in a person who is the subject of epilepsy.

So much, then, for convulsions occurring in the course of ordinary disease. I might also refer to other toxic conditions similar to uræmia in which

Digitized by Google

convulsions occur. There is every reason to suppose that in chronic alcoholism, for example, convulsions occur which depend on the effects of alcohol. We also know that from absinthe much the same effect is produced. By injecting absinthe into animals we can produce convulsions which are very similar to those of epilepsy, and the injection of absinthe into animals has been used very largely for experimental purposes to determine the function of the different parts of the brain.

Convulsions have been defined by Dr. Hughlings Jackson as the sudden occurrence or the simultaneous occurrence of many movements represented in a certain part of the brain, and in associated parts; and one can understand that such a definition would cover not only convulsions which are general, but also convulsions which are special and localised,—that is to say, the convulsions of so-called Jacksonian epilepsy. In the convulsions of Jacksonian epilepsy we have the fit starting in some particular movement and going on to involve other movements, and perhaps to become general. When the convulsion becomes general the particular lesion to which the fit is due is rather apt to be lost sight of. So in considering Jacksonian epilepsy it is well to remember that it ought to be considered to be the condition in which a fit starts in some particular movement, and in which the fit may be actually limited to that one particular movement. Of course, as I have said, you may have the fit spreading from the initial position so as to affect various movements, and, in fact, to affect the whole of the body. Probably the most common form of Jacksonian epilepsy is that in which the fit commences by the movement of the thumb on the index finger; and you may have that movement going on so as to affect the wrist and stopping there, with the hand in the claw-like position; or you may have it going on to affect the whole of the side of the body, or, indeed, the whole of the body. One of the common characteristics of Jacksonian epilepsy is the occurrence of paralysis after the attack has ceased; and the paralysis affects chiefly, and sometimes only, that part of the body in which the fit started. So you may have a fit starting in the hand, and after the fit has passed off you may have only the hand paralysed. Or, on the other hand, you may have the whole arm paralysed. But the chief paralysis will probably be of the fingers in which the fit started. These fits of Jacksonian epilepsy are undoubtedly due to some diseased condition of certain cells or certain centres in the brain; and the question arises whether, in certain cases of Jacksonian epilepsy, you may not have the absence of any real gross disease,—that is to say, whether you may not have in Jacksonian epilepsy a condition extremely like that, or practically indistinguishable from that which we think probably underlies idiopathic epilepsy, as I shall indicate presently when I talk of idiopathic epilepsy. There is, perhaps, very good reason to suppose that in every case of epilepsy, although it is called idiopathic, there is some definite lesion of certain cells in the brain. It may be that this lesion is not discoverable by our present methods; it may consist of some molecular or chemical change in the cells which our present methods are not capable of discovering, and so we must be content in the meantime with theoretical considerations in that respect. But there seems good reason also for believing that in some cases of local epilepsy the pathology is just as obscure as it is in cases of idiopathic epilepsy; indeed, the pathology may be actually the same in the two cases. The reason for this belief is that in many cases of Jacksonian epilepsy the patients go on having their fits just in the same way as ordinary epileptics do, without the development of any evidence of gross disease. Undoubtedly in the great majority of cases of Jacksonian epilepsy there is evidence either of some lesion which has occurred in early life leading to certain local paralysis, such as infantile hemiplegia, or there is evidence of some new growth in the brain giving rise to these attacks. But in certain cases it would seem that there is no evidence of advancing or increasing disease, and the fits of Jacksonian epilepsy themselves are not associated with any paralysis. Jacksonian fits occur in what I have referred to as infantile hemiplegia. Infantile hemiplegia is that form of paralysis which occurs in the first five or six years of life, and is ushered in usually by a convulsion affecting the side subsequently paralysed, or by a series of convulsions affecting that side. After these convulsions have ceased the child is found to be paralysed on the side which has been convulsed, and the paralysis remains permanent, but of course varying in degree in different cases. In some cases the paralysis is

Digitized by Google

very severe, and in other cases the paralysis is extremely slight. Now those patients very often have in after life attacks of epilepsy. They have attacks which are characterised by the occurrence of tonic and clonic convulsions, and by loss of consciousness. In many cases the fit is limited to one side, and in some cases the fits become general. In those cases which I myself have observed, the fits have been restricted to the side which was paralysed; but other observers have described cases in which the fits have been limited to the side which is *not* paralysed. Whether you get the convulsion on one side or on the other, it is usually restricted to one side, but may spread and become general. Those are really cases of Jacksonian epilepsy—that is to say, local epilepsy,—and they are, as we know, associated with some definite lesion in the brain. The nature of the lesion is immaterial, but in this particular case the probability is that there has been some morbid condition of the cortex leading to the abolition of the function of part of the brain, and so to paralysis on one side. Some people say it is stretching a point to call this Jacksonian epilepsy; I do not think it is; they are undoubtedly cases of local epilepsy, and they serve very well to illustrate what is true Jacksonian epilepsy. But probably a more interesting form of Jacksonian epilepsy is that which occurs in association with tumour of the cortex of the brain, and that gives rise to various symptoms. We need not concern ourselves at present with any other than the convulsive seizure; but if a tumour occurs in the Rolandic area, more especially on the surface, particularly if it grows from the membranes, there is irritation of certain parts of the cortex, and in consequence of the irritation of those centres there are certain movements occurring in one or other limb. It depends, of course, on the position of the lesion where you get a convulsive seizure starting. If there is a tumour irritating the hand centre, there is produced a convulsion starting in the hand. If the lesion is near the longitudinal fissure and in the neighbourhood of the leg area, you will probably get a convulsion starting in the leg; and cases have been described in which a convulsion has started in the shoulder, and others in which the convulsion has started in the trunk. It has been suggested by Dr. Hughlings Jackson that the cells of the hand area subserving fine movements may possibly be smaller than those which subserve the larger movements; and one can easily understand that, given an irritant which affects equally large cells and small cells, the small cells will be more likely to discharge first. Consequently, if you can imagine a lesion equally irritating the cells which subserve the trunk movements and those of the fingers, you are more likely to get the discharge starting in the fingers than in the trunk centres. So fits in which the trunk movements are the initial movements are uncommon. The relation of Jacksonian epilepsy to idiopathic epilepsy is very important; and it is important for this reason, that unless you actually see the fit it is very difficult to be sure that in any particular case you are dealing with an ordinary fit of idiopathic epilepsy, or with a Jacksonian convulsion. I remember very well a case I saw in the out-patient department here, which illustrated this difficulty in a very striking way. A man came to the hospital complaining that he had been suffering from fits for about a year. He had had them occasionally, but not at very close intervals, but they were rather a trouble to him, especially in connection with his work. He had headache, but no optic neuritis, he had no local paralysis, and nothing to suggest that his fits were anything but ordinary idiopathic epilepsy; there was no paralysis after the fit, and there was nothing to indicate that the fit was anything but ordinary epilepsy. He was put upon treatment, and remained under it for some months. He improved; the fits remained absent, and in every respect he was better. He went on attending and taking his medicine. One day he came up and then had paralysis of his left arm. On examining his eyes I found he had slight optic neuritis. But there was no headache and no vomiting. I took him into the hospital, and he was in for a considerable time. In the first place, he became very much worse; the paralysis was more extensive, and the leg as well as the arm became affected, and he was in a condition of very marked hemiplegia. He was treated with iodide of potassium and mercury, and after a time he began to improve, and in the course of a few months he went out, apparently perfectly well; his optic neuritis had disappeared, and the paralysis of the arm and leg were gone. About six months later he came back, and was then suffering

Digitized by Google

from very frequent fits. These fits, curiously enough, began in the face, and in many instances were localised to the face, consisting in a drawing up of one side of the face. He was treated in the same way, and again improved and went out; but this time there was a slight degree of weakness remaining on the left side. Although those fits started in the face and affected that chiefly, the limbs on the same side were also affected, and there was some degree of weakness left in them. I think there is little doubt that in that case the patient had a gumma of the dura mater, and the fits at first were so like those of idiopathic epilepsy in the absence of local paralysis and in the occurrence of tongue biting, that one naturally concluded—and I do not think that one could have come to any other conclusion—that the fits were all of an ordinary epileptic character; whereas they afterwards turned out to be true Jacksonian epilepsy, the result of a local lesion in the brain. That illustrates the close relationship that there may be in symptomatology between cases of Jacksonian epilepsy and cases of ordinary idiopathic epilepsy; and I think it is possible that the time may come when we shall no longer distinguish between the two, but look upon Jacksonian epilepsy as epilepsy, and shall also look upon every case of epilepsy as probably a result of some local disease of the brain, *i. e.* Jacksonian epilepsy.

As regards ordinary epilepsy, of course that consists broadly of two great classes—cases of severe epilepsy and cases of slight epilepsy,—in other words, *grand mal* and *petit mal* respectively. As regards *grand mal*, the cases may be either diurnal or nocturnal, or they may occur both during the night and during the day. I think it is very important in every case to find out whether the fits do occur during the night or during the day, or at both times, because, when we come to consider the treatment, I shall have to point out that there is a certain definite line of treatment which I think is extremely useful in cases of nocturnal epilepsy. The prognosis in the night ones, also, is very much better than in the cases in which the attacks occur at all times. With reference to the commencement of the fit, in many instances each fit is ushered in by a definite warning. There are various kinds of auræ or warnings which occur just before the convulsion, and these auræ are the most important thing we have to deal with in cases of epilepsy, because they indicate the part of the brain in which the convulsion starts, *i. e.* the site at which the discharge occurs first. The most common aura is an epigastric aura, which starts with a certain curious undefined feeling in the stomach and usually goes up towards the throat, and very often as soon as the throat is reached the patient loses consciousness. That is by far the most common form of aura in epilepsy. Probably the next most common is the visual aura, one of the special sense auræ. The visual aura may be simply a flash of light, it may be a brilliant coruscation of colours, or it may be a somewhat elaborate visual image. In many cases visual auræ are present, but they are not usually very elaborate. When a visual aura is present in any case of epilepsy, it indicates that the discharging lesion probably is in the visual centre,—that is, in the occipital lobe or in the angular gyrus. There are cases in which there is an auditory aura; it may be a very slight sound or noise, or it may be a ringing noise, or a clanging like a bell. It is not usually elaborated to any great extent, but it is very interesting, because every now and again in such cases a tumour is found to be present. I remember one man who was here some years ago and who had definite symptoms of tumour, with convulsions, and in his case there was an auditory aura. It was usually in the form of a ringing of bells, and for several days before he died he complained of this constant ringing. In that particular case there was a large cystic tumour in the left temporo-sphenoidal lobe. So these cases in which there is a special sense aura are very important, and ought to be investigated very carefully, because they may not be cases of so-called idiopathic epilepsy, but cases in which there is actual local disease of a progressive character, and in which something might perhaps be done for their relief by means of operation. The same is true of the cases in which an olfactory aura is present. A gustatory aura sometimes goes with the olfactory aura, and the gustatory aura may be associated with smacking movements of the lips. In those cases the disease is probably in the temporo-sphenoidal lobe, and in one case in which there was a gustatory aura present there was a tumour in the uncinate gyrus, at the tip of the temporo-sphenoidal lobe. There is another aura, an intellectual aura, or, as others call it, a "dreamy state."

Digitized by Google

In cases in which vertigo is present as the aura, it is difficult to say where the initial lesion may be. But I think it is not improbable that it is in close association with the auditory centre, because we know that the processes of the maintenance of equilibrium are subserved by a branch of the eighth nerve, which supplies the semicircular canals.

There is a very interesting question on which a few words may be said—namely, the determining cause of epileptic fits. I have referred to rickets and the convulsions associated with that disease, and there is no doubt that these convulsions do pave the way for the later occurrence of epilepsy. There is another thing which is very often associated with the onset of the first epileptic fit, and that is a very severe fright. And when one considers the blanching of the face and the extreme disturbance of circulation which occurs in a child when it is very much frightened, it is scarcely surprising that there might be some slight vascular lesion in the brain which might bring on a convulsion; and when a convulsion has once occurred the subsequent occurrence of convulsions is rendered comparatively easy. It seems to be a very simple and easy matter for a convulsion to occur when one has occurred once; it looks as if the path were made smooth for it to occur. And of course what one has to deal with in cases of epilepsy to a great extent is in breaking the vicious habit of the nervous system. However, the occurrence of fright in a child seems not infrequently to determine the onset of the first epileptic fit. Also the influence of the catamenia in women is very important. In many cases of epilepsy the fits occur only at the monthly periods—a day or two before or after, but at all events definitely connected with the catamenia. These are cases which are very apt to be mistaken for cases of hysteria because of this particular connection, but as a matter of fact there is very little doubt that they are true epileptic fits. The effect of pregnancy in epilepsy is very interesting. Many patients who are epileptic have their first fit during pregnancy. On the other hand, many chronic epileptics cease having fits altogether during pregnancy. In others pregnancy does not seem to affect them. So in some cases pregnancy seems to act as a corrective influence in epilepsy, in others to have little or no influence, while in a third class of cases it seems to intensify the severity of the fits, or at all events to increase their frequency. A similar thing is true of acute diseases. When suffering from most acute diseases with a raised temperature, epileptics do not have fits. Patients who are epileptic, if they have an attack of pneumonia with high temperature, during the time that the temperature is up often there is no epileptic attack at all. But as soon as the patient gets into convalescence the attacks are very apt to return. So in the cases I have referred to, in which the patient who is pregnant has no fits, the pregnancy seems to have acted like an acute disease.

Another condition to which I promised in my opening remarks to refer, was the presence of nocturnal fits. In cases where they occur only during the night, it seems to me that the cases are much more tractable by means of drugs than are the cases in which the fits occur during the night and day. For some years I have noticed that in giving those patients digitalis combined with bromide the mixture had a most excellent effect, and this led me to consider whether it might not be the case that these attacks which occur during the night are due to some modification of circulation which occurs during sleep; because patients with "nocturnal" epilepsy often have a fit *during the day if they happen to go to sleep during the day*, so the attacks are not strictly those of nocturnal epilepsy, but are cases of epilepsy occurring *during sleep*. And it seemed to me probable that the good influence of digitalis in these cases may be the result of some modification of circulation which that affects, so preventing the occurrence of attacks which are apt to occur during sleep.

Now a few words with regard to the treatment of ordinary epilepsy. First as to the general considerations. I do not think there is very much in the dietetic treatment of epilepsy, except that one has to be careful that the patient has good wholesome simple food, no food which is difficult to digest or is in any way elaborate. With regard to vegetarianism, I have seen many patients in the out-patient room who became vegetarians on their own account, but I am bound to say I am not very much impressed with the result. As a matter of fact the patients who become vegetarians do not do well; they get into a low condition of general health, and the fits certainly are not modified, either in their frequency or severity, by their dietary. So I have come to regard the best food for epileptics

Digitized by Google

as an ordinary simple mixed diet: one meat meal a day, with plenty of good wholesome simple food. One point which has come up recently is with reference to the withholding of salt from epileptics. The idea is that by forbidding salt you get a better influence from the bromide. In the little experience I have had this does not seem to commend itself to me in any very marked way. The cases which have been treated here on these lines have not done any better than others did who were having salt, so I am not inclined to attach much importance to it, although it has been rather dwelt upon recently as a point in the treatment of epilepsy.

Another point which is very important is with reference to the time at which the food is given. I think no epileptic should have any food within two hours of going to bed, especially if he has nocturnal attacks. Another very important point is that many epileptics have their fits only very early in the morning. I think it is very important to "dodge" those fits, to give the patient a dose of medicine before he or she gets out of bed. It is well to give a small meal consisting of a few mouthfuls of milk and some biscuit, and then let the patient have the medicine immediately after that, and also perhaps half an hour before going to bed. I have known many cases in which the fits occur during dressing, and by means of giving a dose of the medicine before the patient got up one has been able to avert those attacks; and when you are able to avert those attacks in that way you are apt to transfer them to another part of the day. So that it is necessary that you should, so to speak, follow them and attack them whenever they occur, and adapt your therapeutics to the time of occurrence of the fit. But in many of these cases it is effective to give a dose of medicine before the patient gets out of bed, and it sometimes results in a suppression of the fits at all other times of the day as well. The same is true with regard to the other periods at which the fits occur. If the patient has fits habitually at one time, one has to see that the patient has a dose of medicine about an hour before the fits are likely to occur. I am convinced that there is a good deal to be done in the treatment of epilepsy by the observation of those little details. Another point with reference to the food of epileptics is that they must be kept from overeating. Some epileptics are excessively greedy, and are likely to overload their stomachs if they are able. With regard to drugs, of course bromides are the sheet-anchor in epilepsy; and the more I see of the treatment of epilepsy by bromides the more I am encouraged. I do not now see the cases of excessive depression from the use of bromides which I used to see, and that is due to the fact, I believe, that I often combine some drug like nux vomica with bromide in order to prevent it from being depressing. And one rarely gives the bromide of potassium, but nearly always the bromide of sodium or of strontium. The latter is very useful, especially in cases in which the tendency to acne is very marked; and bromide of sodium and bromide of strontium are not so depressing as bromide of potassium. With each of these I nearly always give nux vomica. In many cases one gives digitalis, especially in nocturnal epilepsy,—that is to say, 4 or 5 minims of digitalis with the night dose of bromide of strontium. I tried it fairly effectually, because I gave patients bromides alone first of all, and found the nocturnal fits occurred with that. Afterwards I combined digitalis with the bromide, and the attacks were controlled. Then with regard to another form of bromide salt,—namely, bromide of zinc,—that is very useful, although it can only be given in small doses, because it is an emetic. But in *petit mal*, to which I have made but very little reference, I think bromide of zinc is extremely useful in combination with other bromides. If one could give bromide of zinc in larger doses it would be more useful. With regard to arsenic, that of course one gives only to prevent acne. I do not think arsenic itself has any influence upon the epilepsy; it only has an influence in preventing the acne which the bromide salts are apt to produce. One has to be careful in epilepsy because of the tendency there seems to be towards pigmentation of the skin. It seems to me that arsenic is very much more apt to cause pigmentation when combined with the bromide. I do not know what the reason is—whether it is that the bromide sets up a certain amount of skin irritation which determines the pigmentation by the arsenic,—but the cases of chronic arsenical pigmentation which I have seen in epileptics have been produced by small doses, and they have not been continued over a long time. One certainly sees many cases of arsenical pigmentation in epileptics here, and I have seen it in patients who have only taken small doses of

Digitized by Google

arsenic with bromide for a few months. There is another drug which we ought to use more than we do—namely, borax. But it is not of very much use alone, or only in exceptional cases. I think borax is very useful alone in cases in which the patient has been, so to speak, saturated with bromide. But in chronic cases of epilepsy I think an excellent combination is that of bromide with borax, 10 grains of borax with 15 or 20 of bromide.

With regard to belladonna, I have no very great trust in its efficacy in cases of epilepsy. I have no doubt there are cases of epilepsy in which belladonna is extremely useful, but I cannot myself point out the kind of case in which belladonna does act well. Sometimes when one gives it the results apparently are good, but I cannot say what the indications are for the use of it. With regard to digitalis, I have said it is useful, particularly in nocturnal cases of epilepsy, but I think it is also beneficial in other cases too—namely, those in which the patients have cold and cyanosed extremities. I do not know whether it does good in those cases by improving the circulatory condition, but it certainly is useful.

I may now say a very few words about *petit mal*, those slight attacks of which I have spoken, because they are very important with reference to the diagnosis. Attacks of that form may be of varying character. There may be momentary loss of consciousness, so that the patient's eyes simply become fixed, and nothing else is obvious. There may be a momentary dropping of the patient on his knees suddenly, or he may fall down altogether. Although the attack is momentary, the degree of injury which the patient receives is often considerable. After attacks of *petit mal* there are occasionally conditions of very great medico-legal interest and importance—namely, the condition of automatism, in which the patient carries out elaborate actions and does very curious things in a state of complete unconsciousness. Of course it has been a common defence in cases of murder; it has been stated that the persons charged with the crime have been in an automatic condition of epilepsy. I do not know that it has been an effective defence in a case of murder, but I think it is possible that the patient might in that condition do injury which might result fatally. I have seen a man in the out-patient room here who had an attack of *petit mal*. His wife was with him, and when the automatic condition had supervened on the attack he became rigid and fixed for a minute or two, and after that he became rather violent and tried to knock his wife down. There has been one instance here in which a patient in the out-patient room, after an attack of *petit mal* in the out-patient room, got possession of a poker with which he was about to assault the physician who was seeing him. So the condition is one which is not free from danger, and is one of extreme medico-legal importance. There seems to be very little doubt that in many cases of *petit mal* the patient passes into a condition of unconsciousness, in which he or she does some very elaborate action, and goes long distances and disappears and has to be searched for. There was one case of that kind here which impressed me very much. It was that of a man who lived near London and was subject to epilepsy, and had many epileptic fits in this hospital. He disappeared suddenly some months before, and he told me he remembered being in a certain part of the outskirts of London, and then he knew absolutely nothing until he came to himself in New York. And it transpired he had walked to Liverpool and taken passage and gone to New York, and during that time he had been in a state of mind in which he forgot his previous existence during several weeks, and only came to himself in the streets of New York. As I have already given you to understand, these cases are much more difficult to influence by drugs than are the severe attacks of epilepsy, but the drug which seems to be most useful for *petit mal* is bromide of zinc, of course combined with other bromides. The attacks of *petit mal* are much more important than those of *grand mal*, because of the dangers and complications which may ensue afterwards.

We have received from Messrs. J. S. Fry and Sons, Ltd., some sample tins of a combination of cocoa and milk. The production is prepared from Fry's pure cocoa with sweetened milk from "English pastures." It is justly claimed by the makers that it is a highly concentrated form of nourishment, the milk supplementing the valuable elements present in the pure cocoa. One of the fundamental principles of a real food is the purity of its constituents, and that is secured in this preparation, whilst the necessary attribute of an appetising flavour has not been neglected.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 575. WEDNESDAY, NOVEMBER 4, 1903. Vol. XXIII. No. 3.

CONTENTS.

PAGE

*Thoroughness in Surgery. By W. WATSON CHEYNE, C.B., F.R.S., F.R.C.S. 33

*Post-graduate Lecture on Emphysema and Bronchitis. By FRED. J. SMITH, M.D., F.R.C.P. 43

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

THOROUGHNESS IN SURGERY.*

By W. WATSON CHEYNE, C.B., F.R.S., F.R.C.S.,

Senior Surgeon to King's College Hospital; Professor of Clinical Surgery in King's College.

GENTLEMEN,—The advance in surgery during the last thirty years is one of the most striking points in the general progress of science. Since the introduction of asepsis by Lord Lister there has, it is true, been no further sudden revolution in surgery, but year by year advances have been made in all departments of surgical work, mainly as the result of the protection afforded by this discovery. To those of us who have been engaged in the practice of surgery during the last thirty years, this progress year by year has seemed quite natural and logical, and does not at first sight strike us as anything unexpected or surprising; but if we blot out the intervening period of gradual and steady development, and contrast the state of matters thirty years ago with that now existing, the extraordinary change which has taken place may well make us marvel.

In the first place the amount of operative work now being done is in very striking contrast to that carried on thirty years ago. At that time many large hospitals had only one operating theatre, and operations were only performed on one or two days a week. On these days the theatre was not given up to one individual surgeon, but the various surgeons of the hospital operated in succession according to seniority, the result being that any one surgeon seldom did more than one or two operations. Even under these circumstances the time that the theatre was occupied on each operating day was, as a rule, less than that taken by a single surgeon nowadays. At the present time the

* Delivered at the Torquay Medical Society on October 9th, 1903.

Digitized by Google

amount of operative work has increased to such an extent that in new hospitals and in the reconstruction of many old hospitals an operating theatre is being provided for each surgeon.

Again, the nature of the operative work is very different. The operations which one saw in the operating theatre thirty years ago, and which were looked on as operations of great importance —so-called "major operations,"—now form only a small part of the operative work of a hospital, and many of them are regarded as belonging to the class of minor surgery. When I was a student the ordinary operations that one saw were amputations, the removal of a certain number of tumours, ligature of arteries, operations for stone, stricture, hæmorrhoids, etc., occasionally excisions of joints, or trephining for injuries, opening abscesses, and so forth. At the present time the variety of operative work is very great, and the majority of operations that one sees had not even been thought of thirty years ago, or, had they been thought of, would have been looked on as wild and impracticable.

Another very striking point is that one sees less and less of the old typical operations. Formerly certain definite rules for the various operative precedures were laid down, founded more especially on rapidity of performance and on various anatomical considerations. At the present time operations are more or less atypical. Anatomical considerations must of course always hold their place, but the chief attention is paid not to rapidity or the other matters which concerned the older surgeons, but to the thorough removal of the disease or remedying of the deformity with as little injury to the healthy neighbouring parts as possible.

Again, in former days, in order to have a good supply of cases for teaching purposes, the surgeon required to have a large number of beds, and was very discontented if he only had a few. At the present time, on the other hand, when so much operative work is necessary, the surgeon engaged in private practice finds it impossible to manage a large number of beds; and the tendency is in hospitals, where formerly the number of beds allotted to each surgeon was considerable, to increase the staff so as to make the number of patients under each individual surgeon more manageable.

A further contrast is the kind of cases that one sees in the surgical wards nowadays as compared with those met with formerly. Thirty years ago a comparatively small number of cases in the wards were operation cases. The great majority, such as ulcers, diseases of joints, inflammatory conditions of all sorts, etc., were undergoing various forms of expectant treatment—rest, counter-irritation, and so on. Abdominal cases, which form such a large proportion of those which are now found in the surgical wards, were at that time seldom seen; they were treated in the medical wards, and were only exceptionally referred to the surgeon. On the other hand, at the present time the great majority of the cases in the surgical wards either have been operated on or are waiting for operation, and the more chronic cases, which do not require operation, are comparatively rarely seen, and are, I am afraid, avoided as far as possible. This is an unfortunate circumstance, but nevertheless, I fear, a necessary one, because undoubtedly the object of a general hospital is to do the greatest good to the greatest number, and a chronic case occupying a bed for several months excludes a number of patients who might be benefited by operation, and probably to a greater extent. The result is that many patients who would be much improved by hospital treatment, but who only require rest, counter-irritation, and so forth, are deprived of those advantages; and this has a serious result, not only as regards the patients themselves, but also as regards education, for in private practice cases of this sort form a very considerable proportion of those which are met with, and the student does not have the same opportunity as he formerly had of becoming acquainted with the best methods of dealing with them. The difficulty is so serious that something would need to be done to overcome it. Whether that should be by enlarging existing hospitals and setting apart special wards for chronic cases, by building special hospitals for these cases in suitable situations, or by utilising the Poor Law Infirmaries and affiliating them in some way with the teaching schools, is a question which would need to be considered.

While there is no doubt that this great increase in operative work is of the greatest advantage to the patients, it has its disadvantageous sides as regards the surgeon and the student. One of these we have just alluded to, viz. the absence from the

Digitized by Google

wards of many cases which are of great value for teaching purposes. Another is the tendency for the practice of the operating surgeon to become more of a mechanical art; indeed, it is becoming very much the fashion now to cut short a case by the use of the knife, where perhaps with a little patience and thought the trouble might be overcome in a less severe manner; and this tendency is becoming more and more encouraged by patients who, hearing of rapid recoveries and remarkable successes in many cases, become impatient if they do not themselves recover in a similarly short time. This state of matters is perhaps most marked as regards the question of diagnosis, especially of intra-abdominal troubles; and the large number of exploratory operations performed with the view of diagnosis is quite a new development during this period. There is, I am afraid, a great tendency at the present time to carry this procedure too far, and, on the one hand, to become impatient and make explorations supersede other means of diagnosis; and, on the other hand, to perform explorations for what can do nothing more than merely satisfy curiosity. To explore in all cases where a diagnosis cannot at once be made is undoubtedly a defect in the present system, because it leads to insufficient training of the diagnostic powers, and I think that, unless there is some important reason demanding immediate diagnosis and treatment, one should not explore till other means of diagnosis have been exhausted. An exploratory laparotomy is not always the harmless procedure that is generally supposed. Accidents may happen in the aseptic technique and in other parts of the operation; as, for example, in the separation of existing adhesions, adhesions may form subsequently and give much trouble; and patients who are suffering from debilitating diseases are often the worse for the exploration unless some remedial procedure can be carried out at the same time. For example a patient with inoperable cancer is decidedly not benefited by an exploratory laparotomy. The operation must lead in any case to a certain amount of general disturbance, varying in different cases, partly as the result of the anæsthetic, partly as the result of the intestinal disturbance, partly as the result of the confinement to bed, etc. As a consequence the patient's condition may be very seriously altered for the worse, and, especially in the case of a patient who has been going steadily downhill, is very emaciated, and has only a short time to live, this may be a very serious matter, and may shorten his life to a considerable extent.

I do not wish it to be thought that I am in any way against exploratory operations, but I do think they ought to be employed with great judgment, and not as a mere matter of routine. There are many cases in which, if the diagnosis is doubtful, it is essential to make it certain as soon as possible; in cases, for example, of suspected malignant disease it is important to have an early diagnosis from the point of view of an early radical operation. The rule that I should lay down as regards exploratory operations is that they should only be undertaken where there is a reasonable probability that some remedial procedure can be forthwith carried out,—such procedures, for instance, as the removal of growths, the separation of adhesions, the performance of intestinal anastomosis, and so on. The object of an exploratory laparotomy should not merely be a question of diagnosis; it should be a question of diagnosis in a case where there is a probability of following up the diagnosis immediately by treatment that is likely to benefit the patient.

Now the thing that strikes us most in connection with this great alteration in the amount and character of operative work is that nevertheless the mortality in the surgical wards has markedly decreased, and that a large number of people are restored to health, or their condition very much improved, who would not have been benefited in former days. At the present time, instead of there being comparatively frequent deaths in the surgical wards, one goes through a session with an average of three or four deaths; and such deaths as do occur are, as a rule, the result of the nature of the accident or disease, or are due to shock after operation. Indeed, the great bugbear of operations at the present time is shock, and the next great advance in surgery will, I believe, be in the direction of a better understanding of the nature of this condition, and of the means of preventing or arresting it. If shock could be abolished the area of surgical work would be still further considerably extended.

These considerations lead us to inquire what is at the root of this remarkable change in surgical practice and in the results obtained, and I think

Digitized by Google

that the matter may be summed up in one word, viz. "thoroughness." By using that word I do not, of course, mean to imply that surgeons in former days did not do their work as thoroughly as possible, but that circumstances have so changed that it can be more thoroughly done now. Formerly one of the most important points in performing operations was speed, and a great deal of what we now look on as thoroughness was sacrificed to it. At the present time rapidity in operating is still of great importance, especially from the point of view of shock; and though for a time after the introduction of asepsis rapidity of operating became more and more at a discount, of late the tendency is again to place more value on rapidity so long as the aims of the operation are thoroughly carried out. But, apart from the question of operating dexterity, we have at the present time a much more extensive knowledge of pathology and etiology than was the case formerly; we understand more completely the nature of various diseases, their causation, and the extent to which remedial measures may be adopted; and, under the protection of asepsis, we can apply our knowledge much more thoroughly.

Let us take a few examples of the value of thoroughness. The great example, and the one which lies at the root of the whole modern advance of surgery, is the treatment of wounds, especially as regards asepsis. Asepsis is the very essence of the recent changes in surgery, and the more thorough the asepsis of the wound—that is, the exclusion of bacteria—the more complete and satisfactory are the results which are obtained. I do not propose to go into this question here, as I have written so much about it already, but I may again point out that it is in the actual manipulations in connection with the wound that the essence of the matter consists. Beautiful marble operating theatres, ingenious wash-basins, elaborate steam sterilisers, filtered air, and the various paraphernalia of the present-day operating theatres are all very well, but are quite of secondary importance in comparison with these details.

Some years ago I put the following as a motto in one of my books :—"Suppuration occurring in a wound made by a surgeon through unbroken skin is due to some oversight on his part." This statement holds good now just as much as it did at that time. Where suppuration occurs it is all very well to attribute it to catgut, silk, etc. If it be really due to these causes, it still remains the result of an oversight on the part of the surgeon, for he is responsible that the substances used are properly sterilised. But, as a matter of fact, I believe that it is much more often due to want of thoroughness in the technique of the operation; it is quite easy to sterilise stitches, ligatures, etc., but it is equally easy, unless one is constantly on the watch, to make some oversight in the technique, and thus introduce bacteria into the wound. Of course every one is fallible, and failure may occasionally occur even in the most experienced hands. But the more thoroughly the technique is carried out, the less likely are such accidents to happen. What I want to emphasise is that such failure must not be accepted as a necessity or a probability, but must be looked on as due to some want of thoroughness in the technique employed; and the various procedures must be carefully scrutinised with the view of avoiding such an occurrence in future.

The great result of thoroughness in asepsis is the abolition of septic diseases and the absence of inflammation from wounds. As a consequence we find that various materials, which are in themselves free from bacteria, and which are not mechanically or chemically irritating, may be retained in wounds without causing any trouble, and by their use may aid very greatly in the thoroughness of the operation. For example silver wire, aluminium plates, and various metallic materials may be left in wounds, and are of the greatest value, more especially in operations in connection with bones and joints.

Organic materials may also be similarly employed, and a very important advance at the present time is the use of buried sutures in operating on the soft parts. One is now able to stitch together the deeper parts of wounds and leave the stitches *in situ*. This is a great advantage in many cases, contributing as it does to the thoroughness of the operation, but its success entirely depends on the thoroughness of the asepsis. I know that some surgeons are doubtful as to the value of buried sutures, looking on them as a fruitful source of suppuration and as very likely to be extruded from the wound. But I am satisfied that troubles of that kind are due essentially to want of thoroughness in the asepticity of the procedures. It is true that I have on two or three occasions seen silk

Digitized by Google

sutures come out a considerable time after the operation, although the wound had healed by first intention and no suppuration had taken place at the time. In these cases, after healing had occurred, a little soft prominence formed somewhere or other in the scar, which broke, and through the opening so left a stitch was extruded. This occurrence, however, has been so excessively rare in my experience as not in any way to form a bar to the use of buried sutures; and I believe that even in these instances bacteric infection of the stitch in the first instance is the essence of the trouble, the bacteria buried with the stitch being of a less virulent character than the ordinary pyogenic organisms, though still sufficiently active to interfere with its encapsulation and ultimate absorption.

The value of buried sutures in aseptic wounds is especially marked in such procedures as the repair of the abdominal wall after laparotomy and the radical cure of hernia. My own opinion is that the careful stitching of the deeper layers of the abdominal wall after laparotomy is a point of the very greatest importance. I know that some surgeons do not take this view, and that they are satisfied with a few stitches carried through the whole of the structures of the abdominal wall. I have, however, had to repair quite a number of herniæ which have resulted from this method of stitching up the wound, and, with the experience that buried sutures do not become extruded, I see no reason for running this risk. In laparotomy it is, above all, important to stitch up the peritoneum. I think that one of the great causes of abdominal hernia after these operations is that small portions of the omentum become squeezed out during the after-vomiting, coughing, etc., and, getting in between the surfaces of the wound, prevent the peritoneum and the muscles from coming in contact, and in that way leave a weak spot which is the subsequent point of exit for an abdominal hernia. One sees this very frequently in closing a laparotomy wound, for if the patient is sick or straining or fat, omentum is constantly protruding while the wound is being closed; and it is very difficult to prevent this when only general stitches are used, however tightly they may be put in. It is chiefly with the view of avoiding this protrusion of omentum between the muscular planes that I strongly advocate the closure of the opening in the peritoneum by a continuous suture; but, in addition to that, I also advocate closure of the muscular planes as insuring a stronger repair. I believe that some of the troubles arise from putting these stitches in too tightly, and so pinching up the intervening piece of tissue and leading to its death; if the stitches are only approximated sufficiently to bring the layers into contact, this necrosis does not occur, and they keep the parts together till they have been thoroughly consolidated by the healing process.

The radical cure of inguinal hernia is another striking example of the value of thoroughness in surgery. From being a rare and unsatisfactory operation it has now become one of the most common operations, and is attended with most satisfactory results. The essence of the procedure is, no doubt, complete obliteration of the hernial sac, and in children this is practically all that is necessary; but in the case of large herniæ where the canal is dilated, or in cases where the muscular walls are feeble, the repair of the canal by the use of buried sutures in addition to the closure of the sac is a point of essential importance, and the success of the procedure will depend to a great extent on the thoroughness with which this is carried out.

The imperfect results of radical cure in femoral herniæ as compared with inguinal is an example of the difficulties which arise where the closure of the canal through which the hernia comes is not thoroughly carried out. In femoral hernia, even though the sac be obliterated, the canal through which the hernia came remains large as the result of its previous distension, and the movements of the muscles, instead of tending to close it, as they do in inguinal hernia, rather tend to enlarge it; hence, if something is not done to close the canal, a fresh hernia will almost certainly form.

Various plans have been adopted with the view of closing the femoral canal. The earliest was to stitch back Poupart's ligament to the fascia and periosteum over the ilio-pectineal line, but it was soon found that the result was not satisfactory. The tense Poupart's ligament, pulled on by every movement of the abdomen, very soon tore the stitches out of the fascia and periosteal tissue.

An attempt was then made to carry out the closure more thoroughly by turning up the fascia over the pectineus muscle and forming a diaphragm at the lower end of the canal, but this method also proved to be unsatisfactory.

Some years ago I introduced an operation in

Digitized by Google

which I took a portion of the pectineus muscle itself, as well as the fascia on it, and pulled this portion of tissue up into the canal so as to form a plug in the interior of the canal, the operation being completed by stitching back Poupart's ligament towards the ilio-pectineal line. In a number of instances most satisfactory results have followed this operation. At the same time I must admit that it has not always been successful, and therefore cannot be reckoned as thoroughly satisfactory.

The most recent operation is a recurrence to the original principle of bringing back Poupart's ligament to the ilio-pectineal line. I refer to Roux's operation, in which a steel staple is driven through Poupart's ligament into the innominate bone behind, thus pinning down Poupart's ligament to the bone instead of to the fascia and periosteum, as in the original procedure. The immediate result is most excellent; the canal is completely closed, and if the staple be properly introduced it gets a very firm hold. In only one case have I seen trouble arise from the staple becoming loose, and in it the canal re-opened and the hernia recurred. In that case, however, the staple bent at the time of the operation, and I was not satisfied that it had got a sufficiently satisfactory hold, but I had no other with me. This failure is really an example of want of thoroughness in the procedure. Otherwise the results so far have been excellent. Other plans have been suggested which I have not tried, but the various procedures mentioned are examples of how, at the present time, greater thoroughness in closing the canal is being sought after; and just as the attempts become more thorough so is the success increased.

A very striking example of the value of thoroughness is furnished by the history of laryngectomy and pharyngectomy. In the earlier operations for excision of the larynx the mortality was very considerable—in fact, so great that many surgeons, taking into account also the great physical disability resulting from the operation, considered that it was not a legitimate procedure; at any rate that it was not worth the patient's while to run the very great risk of the operation, considering the bad functional result and the great chance of recurrence.

The great cause of mortality in these cases is septic pneumonia. This infection arises from the entrance of septic material into the lungs, and this may occur either at the time of the operation or at a later period during the after-progress of the case. In the early history of the operation the chief attention was paid to the prevention of the entrance of blood and septic material into the lungs at the time of the operation. These attempts led to the introduction of various methods of blocking the trachea, such as the employment of various cannulæ, notably Hahn's and Trendelenburg's, the insertion of sponges or stuffing above the ordinary tracheotomy tube, operating in the Trendelenburg position, etc.; and by these means the risk of direct infection at the time of the operation is no doubt very much obviated. But nevertheless, in spite of these precautions, septic pneumonia remained a very common occurrence, showing itself frequently from the third to the fifth day; and the reason in all probability was that septic materials passed down into the trachea subsequent to the operation. As formerly carried out, after removal of the larynx a trough was left in the neck with the trachea at the lower and the pharynx at the upper end. This trough was packed with gauze, but it was practically impossible to prevent the septic materials from the wound and the pharynx passing into the opening of the trachea and often infecting the lungs. The continued employment of Hahn's tubes after the operation has been tried, but the great objection to that plan is, in the first place, that the sponge surrounding the tube very soon becomes septic, and in withdrawing it septic fluid is squeezed out and runs down into the lungs; and also that the tube cannot be continued for any length of time on account of the great danger of ulceration and sloughing of the mucous membrance of the trachea as the result of the pressure.

Of late, however, the mortality of the operation from this cause has practically entirely disappeared as the result of a more thorough method of preventing the infection of the lungs. This consists in isolating the divided end of the trachea, stitching it to the skin in such a position that discharges cannot pass into it, and at the same time stitching up the wound above the opening in the trachea, and also the entrance to the pharynx, so as to shut off the throat altogether from the trachea. The result is that discharge does not go down the trachea; and since the introduction of this method the mortality from septic pneumonia has become extremely slight. This is a very

Digitized by Google

striking example of the value of going thoroughly and radically to the root of the matter.

In the case of cancer of the pharynx, for which I have also done a considerable number of operations, the risk is the same or even greater. I have had several very successful cases, but many of the best operations have been defeated by the subsequent occurrence of septic pneumonia, even when the upper orifice of the larynx has not been interfered with. In cases where the disease has spread on to the aryteno-epiglottidean folds, septic pneumonia has generally occurred, in spite of the use of Hahn's cannula during and after the operation, the Trendelenburg position, etc. I have got rather better results since I have tried to stitch up the wound in the pharynx, as can sometimes be done. I think I have seen advantage also, both from the point of view of sepsis and of recurrence, by removing the growth in the pharynx by means of a thermo-cautery in cases where there is no chance of stitching up the wound. In this way the surface is seared, there is less bleeding, there is less discharge for two or three days, just at the time when infection is so very apt to occur; the patient is able to cough and swallow better, and as regards the question of recurrence, the cautery destroys outlying deposits of cancer-cells which may quite well escape the knife. At the same time the results are not perfect, and in future I believe that one must commence the operation by disconnecting the trachea and stitching it to the skin, just as in the case of laryngectomy. It so happens that in a considerable number of cases of cancer of the pharynx and larynx both tubes are infected, and a combined laryngectomy and pharyngectomy may be necessary, and these combined operations are on the whole apparently safer than pharyngectomy alone.

A similar example of thoroughness as regards the avoidance of sepsis is furnished by the history of excision of the rectum. The earliest method of excision of the rectum was the perineal operation, the affected portion of the bowel being clipped out from below, and the upper end in most cases left to discharge fæces and septic material into the raw wound leading to the skin. Under these circumstances septic cellulitis was extremely apt to occur and prove fatal to the patient. The introduction of sacral methods of operation improved matters very much, not only by allowing better access to the disease and thus better removal of the cancerous mass, but also by enabling the end of the rectum to be brought to the skin and stitched there, and so avoiding this long septic tube. Nevertheless the discharges from the bowel very soon infect the wound, and, as a rule, these wounds suppurate and are accompanied by a certain amount of sloughing. With the view of diminishing the soiling of the wound, a good many surgeons advocate a preliminary colotomy so as to cut off the fæcal stream from the perineum; but though no doubt a considerable advantage is gained from this point of view by diverting the fæces, infection still frequently occurs.

Some time ago I began to take still more thorough steps to avoid the infection of the wound, by either closing the lower end of the rectum by means of a ligature, and so preventing the escape of anything from the bowel, or by tying in a Paul's tube. Where the patient is pretty strong I think the most efficient way is, after having brought out the tumour and bowel so that it can reach the skin, to ligature above the tumour in two places, stitch up the wound with a drainage-tube at the anterior part, stitch the muscular coat of the bowel all round to the skin, and then cut away the rectum between the two ligatures, leaving the upper ligature *in situ*. I have had several cases where healing by first intention has occurred practically entirely under those circumstances. The ligature does not separate, and fæces do not appear till the third or fourth day, and the deeper parts of the wound have by that time become fairly firmly healed.

In feeble patients, instead of ligaturing the end of the bowel, I tie in a Paul's tube; but I do not think that this does quite so well—in the first place, because infection is much more likely to happen at the time; and in the second place, because the pressure of the Paul's tube is apt to lead to some ulceration of the bowel. At the same time, in feeble and old people it is not advisable to block the bowel for three or four days, and they will certainly be much more comfortable with a Paul's tube.

Another point with regard to this operation is also an example of the advantage of increased thoroughness. After the sacral operation was introduced it was looked on as practically necessary to make a sacral anus; but it has been found that if the peritoneum is freely divided the upper part of

Digitized by Google

the rectum can be freed very much, so as to come well down, and in a considerable number of cases where the anal portion of the bowel is healthy, the upper and lower portions can be united and the tube restored. But if the two ends of the bowel are stitched together by a circular suture, as is done in the case of other portions of the intestine, it practically always happens that the union gives at some part and a fæcal fistula is established. If, however, instead of making an end-to-end union, the mucous membrane is dissected off the lower portion, and the upper tube is then pulled into the canal so formed, healing may be obtained by first intention with complete restoration of the part.

The improvements in dealing with malignant disease are not limited to the avoidance of sepsis. Marked improvement has also followed as regards the question of recurrence from the increased thoroughness with which the disease has been dealt with of late. Take the case of cancer of the breast. Up till comparatively recently, although the breast was removed, it was quite the rule for the patient to return a few months later with recurrence in the cicatrix, the axilla, and elsewhere, and it was a question with many surgeons whether the operation gave the patient any real prolongation of life. Occasionally cases did occur, however, in which there was a distinct prolongation of life, sometimes lasting over years; but these were the exception, and in the great majority of instances recurrence took place within a year, and death occurred within something like two years. Improvement in the results followed attempts at freer removal of the disease; and if the more thorough operation is done, which I described in 1892, founded on the researches of Stiles and Heidenhain, the results as regards prolongation of life and recurrence are distinctly improved. I have already written several times on this subject, and I am at present looking up my statistics with the view of seeing what sort of final judgment can be come to on the matter. Without referring to any actual numbers, which I have not yet worked out, it is evident that the frequency of local recurrence is much diminished. It is comparatively rarely that a patient comes back with recurrence in the scar or the neighbourhood of the operation; in fact, I do not suppose that I see more than two or three cases of such recurrences in the course of a year. And it is almost equally rare for patients to come back with recurrence in the axilla. Somewhat more frequently recurrences take place in the cervical glands, and it is quite a question whether it might not be advisable to follow Halsted's suggestion and remove fat and glands from the lower part of the neck in all cases, whether they are apparently diseased or not.

What one does find when patients have recurrences is that they take place more especially in internal organs, and sometimes are quite late in appearing. In my last paper I pointed out that up to three years after the operation something like 50 per cent. remained free from recurrence. Recurrence takes place even after that period, but by the older method of operating the great majority of patients were dead of cancer long before three years were completed, so that in that direction there is a very marked prolongation of life. And further, at the present time a very considerable number of cases remain well for many years. Hence the gain from the recent operative procedures is considerable, and this must be put down to greater thoroughness in dealing with the disease.

The modern operation is more thorough than the old one in two ways. In the first place, not only is the local disease removed widely, but all the most likely lines of spread are also taken away. We now know much more than we did before the investigations of Stiles and Heidenhain as to the mode of distribution of cancer of the breast, and the paths by which it spreads; and consequently, by a much freer removal of skin, by the knowledge that the breast extends over a much greater area than we had any idea of formerly, by the removal of all lymphatic channels and glands in the neighbourhood, etc., we make a much wider sweep, and may, and apparently sometimes do, remove the whole of the infected area, with the result that the patient may remain free from recurrence. Even where we cannot get beyond the infected area as regards the body generally, we frequently do so as regards the local part, and, if recurrence takes place, it is in internal organs. As a result the patient lives longer, and ultimately dies with much less suffering than if the disease were allowed to go on in its primary situation.

There is also another, and I believe a very important point of view, which should be considered in this connection. In former days it was not at all uncommon to find that a patient came

Digitized by Google

back a few months after the operation with numerous nodules in the scar and over the whole area of the operation. As the result of this rapid and wide-spread recurrence, some surgeons adopted the view that in many cases the operation lighted up the disease and led to more rapid growth; and, as a consequence, the form of scirrhus called "atrophic scirrhus" was looked on as one which should not be touched. My experience, on the contrary, is that atrophic scirrhus is particularly favourable for operation, and the more the case approaches the atrophic form the greater is the probability of subsequent freedom from the disease. The recurrences referred to are so wide-spread that I think they can hardly be explained on the view that all these deposits existed before the operation and were left behind at the time. The explanation is, I believe, rather in the better technique of the operation at the present time, in that one does not now cut into the actual cancerous deposits. In the old operation, while the main tumour was removed, outlying deposits of cancer-cells were opened up by the knife, and these cells were thus distributed over the wound; and I believe that it was owing to this sowing of the cancer-cells that these very rapid and numerous recurrences were due.

Formerly also, where one was in doubt as to the nature of a tumour of the breast, the plan was to cut into the swelling and inspect it *in situ*, and if it turned out to be a cancer, to proceed to excision. The result of that procedure also was that the cancer-cells were distributed over the part, and rapid recurrence took place. I always lay great stress, in cases where a preliminary examination seems advisable, on removing the lump widely without cutting into it, and on examining it outside the body. Should it then prove to be malignant, all the instruments employed up to that time are discarded, the hands are disinfected, and the wound thoroughly stitched up before the operation is further proceeded with. This, I am satisfied, is no mere theoretical consideration, but a point of the greatest importance in the operative technique of all cases of malignant disease. I have seen several cases which can only be explained on this view, and quite a number of similar cases have been reported.

Take, for example, epitheliomatous glands. It is very common in secondary epitheliomatous glands in the neck for one or more glands to enlarge rapidly, soften, and become semi-fluid; and in some instances these have been opened in mistake for abscesses, with the result that an epitheliomatous ulcer and rapid dissemination of the epithelioma in the cellular tissues has followed. I have on two or three occasions, in excising large masses of glands of this character from the neck, had the misfortune that one of these soft glands has burst, and the fluid has run over the wound. In the first case in which this happened, although I excised the whole mass very freely and afterwards washed out the wound well, and although only quite a small quantity of the material had escaped, I found that after healing had taken place the cicatrix in a very few days became red and thick, and very soon the whole area of the operation was one hard, brawny mass. It was quite evident that the epitheliomatous cells had become implanted all over the raw surface and had infected the wound. Where the glands are very fluid it is exceedingly difficult to avoid this occurrence, so much so that I almost doubt if it is worth while attempting the removal of secondary epitheliomatous glands where marked softening has taken place. In only one case where this accident occurred have I been able to avoid the infection of the wound, and then I not only washed out the wound thoroughly after the operation, but rubbe undiluted carbolic acid over the whole surface.

The tendency to greater thoroughness in cases of malignant disease is not limited to the breast. For instance, in cancer of the tongue some surgeons strongly advocate that the anterior triangle of the neck should be cleared out at the same time, whether any noticeable glands are felt or not, just as is done in the case of the axilla. This is a view which I think is open to debate, and personally, in these cases I prefer to feel one or two enlarged glands before clearing out the anterior triangle. I think that the removal of the fat and glands is much better effected if one has a little guide in the shape of some enlargement of the glands. I have always found it excessively difficult to clear out the anterior triangle satisfactorily where no enlarged glands were present; and a further objection that I have to this procedure is that if glands are left behind and subsequently enlarge, the cicatricial tissue from the former operation renders any thorough removal of the glands afterwards an excessively difficult and almost impossible matter.

Digitized by Google

Tuberculosis is another example of a disease in which the benefit of greater thoroughness is very markedly evident. Formerly surgical tuberculoses were, as a rule, left to Nature to cure, assisted by rest, good food, and so forth. Abscesses were opened, and might or might not heal, and operations, if performed, were very often imperfect as far as removal of disease was concerned, and much of the tuberculous tissue was left behind. For example, in the original operation of excision of joints the incisions were made on certain anatomical lines, with the result that practically the whole of the synovial membrane was left behind; and, naturally enough, recurrences very often took place, sinuses remained, and in many cases amputation had ultimately to be performed. Nowadays we know a great deal more about the pathology of tubeculous lesions, and have learned, on the one hand, that much more thorough removal is necessary; and, on the other, that in an excision done on the old lines a larger amount of healthy tissue is taken away than is always necessary. In children such an operation as excision of joints, especially of the knee-joint, is not as a rule requisite or advisable, and the removal of the tuberculous tissue alone can be very much more thoroughly carried out with much less risk of interference with the growing parts of the bone by arthrectomy than if the old operation of excision is performed.

It has also been recognised that in the case of a tuberculous abscess it is not the fluid contents, but the wall of the abscess which is the important part, and that the latter must be thoroughly removed or dealt with in some way if rapid healing is to be obtained.

A very important subject is the treatment of tuberculous glands, and there the result of the operation depends essentially on the thoroughness with which it is done. The removal of individual enlarged glands from the triangles of the neck is, as a rule, quite futile. The operation must consist not only in removal of the visibly enlarged glands, but of the whole lymphatic area. One often sees cases of softening glands in which the patient is under the impression that the removal of the enlarged glands is all that is necessary, and that it is a comparatively small operation. Now in the case of glands in the neck the operation never is a small one, and, although only a few glands may be enlarged, the operation must consist in the removal of the whole glandular area; otherwise the result will almost inevitably be disappointing. If, however, the removal is carried out thoroughly, and the whole infected lymphatic area is taken away, I look on the operation as an extremely satisfactory one; but to be satisfactory it must be thorough on these lines, and the fact that some surgeons are giving up excision of glands in the neck depends, I believe, on want of thoroughness as regards the extent of the removal. In many cases this want of thoroughness is due, again, to want of thoroughness in the aseptic technique, the result being that the surgeon fears an extensive operation like that necessary for completely clearing the glandular area on account of the risk of suppuration in the deeper planes of the neck. I consider that, with proper aseptic technique and with wide removal not only of the visible disease, but of the paths along which the disease travels, operation for the removal of tuberculous glands is an exceedingly satisfactory procedure.

There are many other examples of thoroughness which I might mention. In fractures, for example, why should we be content nowadays with an imperfect result? If the broken ends are not in good position, if we find that apparatus of various kinds will not keep them in good position, why should we wait and be content with the best result that we can get by these means? In some cases, of course, it may be advisable to do so, but in others, instead of applying pads, etc., to keep the bones in position, and failing as one so constantly does, I see no reason why we should not intervene by suitable pegging, screwing, or other methods of fixation of the fragments so as to obtain a perfect result. It is a pure question of carpentry. I do not say that operative interference need be carried out in a large proportion of cases of fracture, but I have seen many cases in which much better results would have been obtained by operation than resulted from the use of apparatus. In fractures of the patella and olecranon I think there can be no question that operation is the proper procedure unless under exceptional circumstances; and in many fractures about the tibia, about the elbow, fractures of both bones of the arm where the bones tend to come together, etc., much better results will be obtained by operation than by any apparatus.

Digitized by Google

The whole range of abdominal surgery also shows the value of thoroughness. The operative work on the stomach, notably for ulcer of the stomach, is a very striking example. The various operations on the gall-bladder, appendix, intestine, kidney and ureter, prostate, etc., are all recent developments resulting from more thorough consideration of the best methods of alleviating or curing a variety of conditions. In the case of the thorax also, though the difficulties are greater, the advances are marked. To take one small example, viz. empyema, we know what a long time it may be before healing occurs if the older plan of simply opening the thorax and putting in a drainage-tube be adopted. On the other hand, in suitable cases, more especially in children, if, instead of putting in a drainage-tube at once, one allows the patient to wake up partially and excites coughing till the lung has completely expanded, healing often occurs very rapidly.

I might go on for hours indicating the improvements which have been made of late years in surgical work, but I think I have said enough to show that they have resulted from greater thoroughness on the one hand in the elucidation of scientific knowledge, and its application to practice, and on the other, in the actual technique of operations. The old need for hurry is past, as is also a great deal of the anxiety as regards the septic risks. With the patient unconscious and in the hands of a competent anæsthetist, the fear of causing pain and the struggles of the victim have gone, and the need for lightning speed, which must have interfered greatly with thoroughness, has disappeared. The anxiety as regards sepsis has also diminished, and with experience and more especially with a previous thorough bacteriological training, the aseptic technique becomes automatic and does not distract the surgeon from the objects of the operation, viz. thorough dealing with the disease. The surgeon may therefore, at the present time, direct his whole attention to the problem before him, viz. the best way of remedying the trouble; and practically the only limits to his activity are anatomical considerations, the existence of complications, and the question of shock. His great motto must be "thoroughness," and every case which does not turn out satisfactorily must be carefully criticised to see in what direction the treatment could have been more thoroughly carried out. Slipshod surgery, doing a thing "well enough," "removing as much of the disease as possible" (a most pernicious procedure, especially in the case of malignant disease, for the reasons already stated) are to be most strongly deprecated. Of course one cannot do impossibilities, and there are many cases in which ideal thoroughness is impracticable; but still there are many where one fails to obtain the best result, and where, on thinking it over, one may find room for improvement. I think that this should be the frame of mind in which a surgeon should approach his work, and that the old copy-book heading applies very strongly to surgical work—namely, "What is worth doing at all is worth doing well."

POST-GRADUATE LECTURE

ON

EMPHYSEMA AND BRONCHITIS.

By FRED. J. SMITH, M.D., F.R.C.P.,

Physician to the London Hospital.

Delivered at the Polyclinic, October 6th, 1903.

GENTLEMEN,—The audiences which I am in the habit of addressing in my lectures usually consist of students who are not yet qualified; and I would ask your kind indulgence, therefore, if I tell you some things which may seem a little commonplace, and about which you probably know as much as I do. I would, however, remind you that the commoner a disease is the more likely one is to pass it by as not worth any further study, a mental attitude which I am sure is likely to be productive of considerable mischief, and possibly even of serious damage to that reputation by which a medical man maintains his practice. I think, therefore, I need make no further excuse for talking to you on one of the commonest of diseases, viz. emphysema and bronchitis, with their complications and treatment.

A fairly prolonged and extensive experience in the post-mortem room and out-patient department has convinced me that when once the age of 40 has appeared some degree of emphysema is one of the commonest, if not the absolutely commonest, condition in the lungs of every one; and I have no

Digitized by Google

hesitation in saying that every year thousands of deaths in adults are caused by emphysema in association with its attendant troubles of bronchitis, broncho-pneumonia, and heart failure. On the other hand, I have very rarely, if ever, found an emphysematous condition of the lungs (uncomplicated by other troubles, such as phthisis, or fibroid lung, or possibly cases of congenital asthma) after death in a young subject; and it is very rare indeed to find the condition during life so marked as to be recognisable in children whose lungs are otherwise healthy. Even after prolonged illness from whooping-cough I could never satisfy myself that permanent mischief had been done to the lungs by that disease alone (uncomplicated with tubercle), though, as we might naturally expect, the organs are found to be incapable of standing such a prolonged strain as is involved in chronic asthma which has persisted for years; but this is happily a rare condition, probably dependent on some hereditary influence, and possibly associated with an originally indifferent quality of the elastic tissue of the lung.

This peculiarity of age-incidence is, I am convinced, very marked (two years' statistics of cases of bronchitis and emphysema at the London Hospital give me 113 males to 46 females, with an average age of 48·6 for the males and 46·7 for the females), and has, I think, an exceedingly strong bearing on the pathology of the disease, which I will now proceed to sketch for you, with its morbid anatomy, the latter representing the terminal results of the former.

Morbid anatomy.—Within the last year I have seen two of the most extraordinary cases I can recollect, two in which the lungs on removal looked like a collection of large blisters from apex to base; the condition existed over the whole surface. Omitting, however, such very extreme cases, we find that the lung on removal looks big and *pale* (the pallor is a very marked feature) all over—when the process is at all extensive, but in less marked cases it is only the apices and margins that have this appearance, the general surface being either unaltered or marked with indentations corresponding to the ribs; the bigness is, however, deceptive, for on applying very slight pressure a deep pitting or collapse of the structure takes place, and the mark thus made does not disappear if left alone, though it can be very readily made to do so by squeezing air in from a neighbouring area. The feel to the pinching fingers is peculiarly soft and downy, like silk, and one misses very much the crepitations usually to be perceived on pinching a piece of lung. On section of the lung, pus can generally be squeezed from the smaller tubes if the patient have succumbed to an illness of pulmonary or cardiac type, but nothing is commoner in advanced or middle age than to find the edges and apices emphysematous when the symptoms of the fatal illness had never suggested such a condition; this is a point of great clinical importance. If pus can thus be squeezed out, a little closer inspection is almost sure to reveal areas of consolidation—broncho-pneumonia, in fact,—and also areas of collapse, the latter recognisable by their colour being darker than the general colour of the lung, and by their sunken position below the general level, quite apart from the bulging of the emphysematous areas. Small hæmorrhagic foci are also not uncommon, though marked solidification from hæmorrhage is a condition I have seen only once or twice (one very remarkable case of the kind occurred to me many years ago, in which there were many dozens of such hæmorrhagically consolidated areas, strictly the size of marbles, in that they varied as marbles do). The condition known as bronchiectasis, I am inclined to think, is comparatively rare, unless there is a gross amount of fibrotic thickening, in which case the emphysema is probably quite a secondary matter; and the chronic inflammatory condition demands all one's clinical attention—a condition I do not propose to touch upon to-day, nor do I wish to more than mention the so-called compensatory emphysema round tubercular or fibroid patches.

In cases of marked emphysema the heart is rarely in a healthy condition; the right side of it is commonly increased in the volume of its chambers, and generally this has happened without a proportionate increase in the thickness of the muscle which has to empty those chambers; in fact, it is the muscle which attracts attention. There are three naked-eye changes very common: (1) a great increase in the gross fatty covering of the right ventricle, with apparent thinning of the muscle substance (I say apparent because, carefully as I have cut many of these ventricles, I am convinced that I must have misunderstood the amount and thickness of the muscle, for no heart could possibly

Digitized by Google

continue beating without some muscle, and it has frequently happened to me to be unable to see any at all except perhaps the thinnest of strips, very pale indeed, and looking fatty); (2) a condition of brown atrophy, in which the heart muscle looks very much browner than usual, and unhealthy; (3) typical fatty degeneration of the whole heart muscle with definite tabby-cat striation.

Such are the naked-eye appearances, and the microscope only completes the details and explains the meaning of what we see and feel. Thus in the lung the alveolar spaces are found to be enormously increased in superficial area, with a corresponding thinness of the walls; the septa between the spaces are found atrophied and broken; and the elastic tissue fragmentary and frayed. Thousands of such septa also must have entirely disappeared, so that (1) an enormous amount of the original aërating surface of the lung must have disappeared, (2) the obstacle to forcing blood through the remaining stretched capillaries must be very much increased; and (3) the actual aërating surface which remains is not so well adapted for the purpose as that originally present, for we find microscopically that the mucous membrane of the smaller bronchioles is unhealthy: the epithelium is irregular in disposition; it has often quite disappeared, and may be now and again found quite loose in the lumen—in fact, a catarrhal process has been going on. The smaller blood-vessels also very frequently show some thickening of their walls, and especially of the intima—a condition of atheroma, in fact,—and so constant is this condition of the vessels that I regard it myself as one of the strongest arguments in favour of the view which I adopt as to the pathology of the disease. The hæmorrhagic foci when present show the usual appearances of small blood extravasations, with changes in the lung tissue similar to those described above. The heart muscle shows changes of fatty degeneration, with loss of good striation. But I am not concerned to labour the microscopic changes, and I have said enough to enable one to understand the pathology or the morbid physiology by which these changes are produced.

Now to a special pathologist studying dead results it is of the utmost interest and importance to know what particular form of degeneration is at work—fatty, colloid, granular, mucoid, etc.,—but to the physician who has to consider organs from the point of view of their capability of performing the function for which they were built, the fact itself of a degeneration is the important point, involving as it must a loss of vitality. The exact nature of the change which has produced the loss is comparatively immaterial; all the microscopic changes above detailed indicate a loss of vital function in the structures enumerated, and it is by this loss that symptoms are produced.

Hence I hold very strongly to the view that the question which has been so hotly debated whether it is during inspiration or expiration that emphysema is produced is of quite secondary importance, and that the important one is how or why did the tissues whose business it is to see that emphysema does not arise, how or why did they degenerate. A piece of rotten elastic will break when being actively stretched, as in inspiration, equally easily as when it is trying to recoil against pressure, as in expiration, though as a matter of fact I believe in the expiratory view; anyhow, violent cough is the active factor.

No; emphysema has got to take its place as a term in the very ill-understood expansion of the function vitality. Scientific precision of expression of detailed facts has, I am sure, one great defect in that it is apt to obscure the general principle that no living, nor for that matter no dead thing, can last for ever and do work. It must wear out sooner or later. If the latter we call it natural and general senility; if the former we speak of it as special or premature senility of some organ or tissue; and emphysema is senility of lung tissues, whether natural or premature. Often the former, and associated with all other signs and symptoms of senility, but frequently enough the latter, and then it requires to be diagnosed and its presence remembered as a sign of age with important bearings on prognosis.

Diagnosis.—The pathology which I have thus briefly sketched for you is of great and practical importance in diagnosis, as well as in treatment and prognosis. I need hardly remind you of the typical text-book case with barrel-shaped, almost immobile chest, bowed back, and a racking cough, especially in the morning, and which has been bad in winter and better in summer for many years. This is the well-marked case to be seen at a glance, but his symptoms, exclusive of the cough, which is the associated bronchitis, are worth a little study that

Digitized by Google

we may be able to detect the trouble in an early stage and possibly help to arrest its progress.

Shortness of breath is his first and great complaint. Now shortness of breath obviously means a disproportion between the amount of oxygen required by the body and the available functionally capable aërating surface (note the functionally capable). The reduction may be due to (1) absolute diminution though the balance be healthy; (2) to unhealthy condition of a normal area due to undue secretion in the tubes, either (*a*) epithelial catarrh or (*b*) excessive exudation; (3) (very closely allied with 2 *b*) imperfect circulation through the lungs. Hence, though perhaps the process is so rapid we are unconscious of it, a diagnosis of emphysema is really one of exclusion, *i. e.* exclusion of other causes than emphysema for the shortness of breath.

1. Diminution in area has for its prominent causations (*a*) collapse from fluid or from solid growth, both of which are rapidly excluded by there being no dulness and increased resistance on percussion, and no ægophony nor bronchophony; (*b*) collapse from blockage of tubes rapidly suspected by the local absence of breath-sounds with many adventitious ones elsewhere; (*c*) consolidations recognised by tubular breathing, etc.

2. Unhealthy conditions of a normal area have for their associates rhonchi and crepitations, caused either by a coincident bronchitis, in which case, though we feel pretty sure in an elderly patient that emphysema is present, its establishment requires other considerations, principally of disproportion between the shortness of breath and the rhonchi; or they are caused by circulatory disturbances which we recognise by departures from health in the sounds of the heart (not necessarily bruits).

3 *Disturbances in circulation.*—These are almost invariably associated with fine crepitations, especially at one or both bases; and again, the heart-sounds, bruits, and relative lengths of the first and second sounds and the frequency of the beat require most careful estimation.

Hæmoptysis.—The occasional occurrence of this symptom is fully explained by the morbid anatomy of emphysema. Stress must be laid on its occcurrence, because of all symptoms it is the one which suggests the presence of tubercle; and when it has occurred special pains must be taken to exclude phthisis by making repeated examinations of the sputum for bacilli. I find in the 'London Hospital Records' it is mentioned in about 16 per cent. of cases.

Alterations in percussion.—These constitute by far the most important of the physical signs of the disease. When it is present the cardiac dulness is diminished in proportion to the degree of emphysema present, not beginning till the fifth or even sixth cartilage on the left side; while the hepatic dulness may be inappreciable till the seventh or eighth rib on the right is percussed.

Auscultation shows an absence or diminution of the natural murmur over areas where the percussion note proves that lung tissue still lies beneath the thoracic parietes.

Such are the principal scientific data upon which to verify a diagnosis; but I think one's clinical experience often leads one into making a short cut. One knows that a chronic cough is invariably in a person past fifty years of age associated with some degree of emphysema. I have very frequently found that the above physical signs are well marked in these elderly patients when suffering from a slight bronchitis, which they confidently assert is the very first attack they have ever had. They entirely deny having ever had a cough previously, and though they may at first have forgotten such a cough, I believe that cross-examination would recall the fact; at least, I know it would in my own case, and personal experience in milder diseases is most valuable. It is facts such as these that compel one clinically to regard emphysema as essentially a degenerative or senile process, not necessarily caused by bronchitis, but made more manifest and probably also much worse by catarrhal attacks. In brief, the relation between bronchitis and emphysema, in my opinion, is as follows:—First, slight emphysema arising from weakened nutrition predisposes to bronchitis, and this in turn leads to strain (both expiratory and inspiratory) on weakened tissues, and this strain increases the emphysema and so establishes a vicious circle, the chief outward sign of which is a *shortness of breath, for which no very obvious coarse lesion is present to account.*

Prognosis.—Diagnosis, whether simple or elaborate, is not of much use unless it helps us to a better comprehension of the whole case; and the point I wish to emphasise in this connection is that we must be very cautious in promising too much to

Digitized by Google

a patient over forty-five whom we are called upon to attend with what at first sight seems a simple cold or slight laryngeal catarrh. My own personal experience is that a bad cold in the nose is pretty sure, in winter at least, to be followed by a nasty laryngitis, and that in turn by a cough of greater or less severity for some weeks; and if with this there are any physical signs of emphysema, the prognosis of *complete* recovery is bad—recovery, that is, which brings the patient back to a *statu quo antea.*

Owing to associated emphysema I always look with suspicion on broncho-pneumonia in an elderly patient. It is true that I rarely find in the post-mortem room a death stated to be solely and entirely due to bronchitis and emphysema in adults; but that the latter at any rate is a seriously complicating factor I have equally very little doubt.

But it is perhaps not as threatening life itself that it is so important to recognise the presence of emphysema, but as threatening comfort and capability of accommodating one's self to one's environment; the blood-stained or streaked sputum, too, will keep the patient—aye, and even the doctor—on the tenterhooks of threatening consumption, and cause much worry; while the shortness of breath, for which we can do little or nothing, will cripple a working man and annoy a richer patient.

Treatment.—It is a mere truism to say that treatment to be genuinely successful should be based on pathology. Now the points I have endeavoured to establish in the pathology of the trouble we are now discussing are—(1) that it is essentially a senile change; (2) that it causes very great liability to bronchitis and inflammatory exudations; (3) that bronchitis and inflammations tend to make it much worse; and (4) that the heart suffers very much in proportion to the degree of emphysema present.

With regard to (1) we cannot, perhaps, take any very active steps to retard it, but we may possibly help by advice; one of the principal items of which is that we should dissuade our elderly patients from undertaking any very strenuous physical labour which will cause the respiratory movements to become unduly deep. I have not the slightest doubt but that emphysema is the real meaning of the fact that labourers—*i. e.* navvies, blacksmiths, and all those whose work is really physically laborious—are rarely found to be capable of doing a full day's work much past the age of, say, forty-five to fifty; or, if so, they have to go more slowly at it than they used to do; "go slow" must, in fact, be our motto for our elderly friends if they would not hurry down the decline of life.

I could add a few words on climate, but think that might lead one too far, but there is no doubt about a warm dry climate being best for those who can afford it.

2. The liability to catarrh is somewhat intimately bound up with exertion in that this leads to free perspiration, and perspiration naturally cools the skin; and it is impossible for us to draw the line of distinction between a pleasant cooling and one that is dangerous to health; in fact there is none. Nor do we understand how it is that we can sometimes cool ourselves at an open window without the slightest harm and sometimes not; I am sure it is a risk over forty-five. There is another point, too, in this perspiration which I am sure is important—at any rate in my own personal experience,—but which is usually entirely overlooked, and that is damp, *not wet* feet. After actual wading in water, or when the boots are soaked through from wet grass, etc., the boots and stockings are changed readily enough, but when no such obvious wetting is in question the stockings at any rate are often left unchanged. Now the feet perspire quite as readily as any other part of the body, and owing to the general system of ventilation in use in England under doors, etc., they are very frequently kept for hours in draughts while one is sitting after outdoor exercise. From my own experience I am quite convinced that this is the real reason why we catch cold when sitting in hot, close, confined rooms in the evening, where air is only admitted under the crack of the doorway, and is drawn along the floor to an open fireplace. The factor of change of temperature is an important one, too, owing to the cough which it frequently sets up, the violence of the necessary respiratory movements being detrimental to the failing elastic tissue of the lungs. This, and not any special peculiarity in night air, beyond the fact that it is probably cooler than the air of sitting-rooms at night, is the reason why we ought to advise elderly patients not to go out much at night. Smoking must be judged by its effects; I do not believe that the act itself is necessarily and always injurious, but it is if it excites coughing. I have learnt a little trick by experience which I think is worth mentioning; it is when coughing to forcibly hold the arms into the sides so

Digitized by Google

as to assist by compression the expiratory forces. I have found it helps me to clear the bronchi with less violence and less prolongation of the cough, both of which are points well worth bearing in mind.

Should our patient have unfortunately got an attack of bronchitis, it is obviously our object to cure him as speedily as possible, for each attack, and especially if prolonged, leaves some scars behind it. I have nothing new to add to what you all know as to the treatment of bronchitis, unless it be as to the great use I have found in an old-fashioned remedy, viz. syrup of tolu, and also in one new one, viz. creasote. It is usually assumed that this latter is good for tubercle only (by the way, in my opinion it comes for tubercle as near to a specific as we are likely to get, unless through some anti-serum discovery), but I have tried it in many cases in which I did not suspect tubercle, as well as in those in which I did, and I have found it of the greatest possible benefit in cases that were apparently simple catarrh only, especially in combination with cod liver oil. Another point is this: if our patient is in the habit of having a cold bath in the morning, don't stop it; it is in itself a healthy habit, and in a bronchial attack assists most materially in clearing the tubes.

Lastly, we have to consider the treatment of the heart in emphysema, whether this be simple or complicated by catarrh; and upon this subject I have one or two remarks to make which are by no means new, but which are so important they will well bear repetition and emphasis. A word or two on bleeding, for instance. I am convinced that it has gone too much out of fashion, and I am sure that I have saved several lives, temporarily at any rate, by a timely bleeding from a vein. It will not, of course, *cure* either the emphysema or the heart condition, but if matters have not gone too far it will restore a balance between the heart and its work which has been lost, and it may be that the balance may remain adjusted for quite a long interval. Blood must, when necessary, be taken freely, *i. e.* not less than ten ounces from an adult, and better the nearer to twenty ounces we can go. In my opinion the necessity is measured by two factors—(1) the blueness of the lips and nose, and (2) the breathlessness of the patient. It makes one feel as happy as a surgeon after a successful operation to see the relief rapidly stealing over the patient as the blood flows more and more freely. Then the use of opium and morphia in heart troubles. It is well worth bearing in mind that morphia is one of the most, if not the most valuable cardiac tonic we possess. It may be that it is not its true pharmacological effects upon the heart itself that is of value, though of this there is a good deal of experimental evidence when used in smallish doses; but much more important is the sleep it induces in most cases. Insomnia is one of the most distressing features of cardiac trouble, and a night's quiet sleep will do more to restore the body than anything that I know of; and if, say, $\frac{1}{8}$ grain morphia be injected, it rarely fails to give sleep. I admit that heroic doses are not advisable, but I cannot make out whence comes the dread of morphia in cardiac distress that I find so universally entertained by practitioners. Lastly, let me add a few practical words on digitalis, which of all the list of direct cardiac drugs—convallaria, strophanthus, scopariine, etc., etc.—seems to be the reliable one, and one that maintains its position through the changing fashions. I am sure by my own experience that it is usually given in too large a dose; if 3 or 4 minims of the tincture, or 1 drachm of the infusion, or 1 grain of the powdered leaves do not help us, I have never found it of the slightest use to increase the dose, and with larger doses the alleged cumulative action is more likely to appear. I say alleged because when one knows that digitalis is a drug that is moderately quickly absorbed and very slowly excreted by the urine, then I say that he who gives digitalis and pushes it without noticing the daily quantity of urine is at fault, and not the drug he is carelessly using.

I must thank you, gentlemen, for listening to me with attention while wearying you with such commonplaces, but I can only hope I have given you some food for reflection, and that you may feel a little happier in the next case of emphysema you have to manage.

THE action of sparteine sulphate is comparable to that of digitalis, and it sometimes succeeds when that drug fails. For some years "Tabloid" Hypodermic Sparteine Sulphate gr. $\frac{1}{3}$ has been issued, but to meet the requirements of those who wish to administer the drug by the mouth a "tabloid" product containing gr. 1 is now prepared by Messrs. Burroughs Wellcome & Co.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 576. WEDNESDAY, NOVEMBER 11, 1903. Vol. XXIII. No. 4.

CONTENTS.

PAGE

*A Clinical Lecture on Some Points Relating to Rigidity of the Spine. By Sir WILLIAM BENNETT, K.C.V.O., F.R.C.S. 49

*A Clinical Lecture on Some Common Nerve Injuries: their Examination and Diagnosis. By JAMES SHERREN, F.R.C.S. 53

*Large Mononuclear Leucocytosis in Malaria, Blackwater Fever, and Lymphadenoma. By FRANK BUSHNELL, M.D. 59

** Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE
ON
SOME POINTS RELATING TO RIGIDITY OF THE SPINE.

Delivered at St. George's Hospital.

By SIR WILLIAM BENNETT, K.C.V.O., F.R.C.S.

Surgeon to the Hospital.

GENTLEMEN,—I have often warned you in these lectures against attaching too much importance to any one sign in the diagnosis of disease,—in other words, against accepting singly, as final, any of the signs which are spoken of as characteristic or pathognomonic in the text-books. Speaking generally, there is no one sign, however precise it may seem to be, which is certain evidence of any disease unless it is corroborated by other symptoms or signs. There is a habit with many people of speaking of rigidity of the spinal column as if it were positive evidence of organic disease of the part. Now, although it is true that stiffness of the spine is, in the majority of cases, fair presumptive evidence of disease either in the vertebral column itself or in the parts immediately around it, this symptom is not always evidence of disease of the spine. There are, in fact, two kinds of rigidity of the spine, one of which is evidence of disease and the other which may exist independently of organic lesion. These two varieties differ a little in their clinical aspects. There is a stiffness of the spine which is absolute, and there is a stiffness which is hesitating or stammering. The difference in the two conditions should be apparent without any explanation. In the absolute stiffness the whole spine, or a portion of it, is as rigid as a piece of wood. In the other kind—the hesitating or stammering rigidity,—as the patient attempts to bend the spine, although at first the column appears to be quite stiff, as the attempt at bending is con-

Digitized by Google

tinued you will find that what at first seemed to be complete rigidity becomes merely a kind of hesitation or stammering; the muscles, in fact, seem reluctantly to allow the bending to take place by a number of small and succesive jerks. This stammering in the flexion of the spinal column is important, and not commonly understood. If you are unaware of its existence you may under certain circumstances be deceived, and be led to think that organic disease is present when it does not exist. In an adult the examination of the spine in order to detect disease is, as you know, simple enough. It is easy to stand the patient before you and to tell him or her, as the case may be, to bend forward as far as practicable, and then to resume the vertical position, whilst you keep one or two hands placed upon the spinous processes so that you may detect whether the individual vertebral joints move or not. In infants and very young children it is not always a simple matter, unless you know exactly how to arrive at it, to make sure whether the spine is or whether it is not stiff. It is not very uncommon to see practitioners, whose position would lead one to expect more discrimination, stand small children up before them and tell the little things to stoop and bend, etc., with a view to testing the movements of the spinal column—a proceeding which obtains at the best very deceptive information, because the subjects submitted to the test are not educated sufficiently to carry out with intelligence the required movements. In examining young children and infants for rigidity of the spine the proper plan is not to have the patients standing up, but to place them lying flat on the back on a bed, couch, or table. If one hand of the surgeon or practitioner be now placed beneath the spine and an attempt made to raise the patient, it will be found, if the spine is rigid, that it does not "give" before the hand at all, but moves as a solid mass or bar.

If, on the other hand, there is no rigidity, or only partial rigidity of the stammering kind, you will find that as the spinal column is raised by the hand it bends before it. This is the most delicate test—and, indeed, the only truly reliable one—for determining in a young subject whether the spine is rigid or whether it is not; the ordinary tests which are employed for adult people are very fallacious in young subjects, a fact the knowledge of which is of some value, and may obviate mistakes in diagnosis sometimes of much moment. Under the influence of an anæsthetic rigidity of the spine may disappear or it may not; I am now speaking of rigidity which is undoubted. In a general way, if the rigidity disappears under the influence of an anæsthetic, you may consider that there is probably no very grave disease present. But the fact that the rigidity disappears upon the administration of an anæsthetic is no certain sign that disease is absent, because you must bear in mind that in the case of the spinal column, as in any other joint or joints, the spine being of course nothing more than a very complicated and long articulation, in the early stage of inflammatory conditions in the articulations, whilst the patient is conscious and susceptible of pain there will always be rigidity, in consequence of the protective action of the surrounding muscles; when an anæsthetic has made the patient unconscious of pain, the necessity for protective rigidity of the muscles no longer exists, and you may have, in a case in which there is real disease, an entire disappearance of rigidity existing when the patient is conscious. On the other hand, supposing the rigidity still persists under the anæsthesia, it is not invariably evidence of the existence of organic disease. It is well to bear in mind that in some cases the examination of a patient under the influence of an anæsthetic is not a help to diagnosis, but the reverse, because the anæsthetic may lead to the disappearance, for the time being, of symptoms due to disease.

I have already said that rigidity of the spine may or may not be the result of organic disease. Rigidity of the spine may involve the whole length of the column, or it may affect only a limited portion. Absolute rigidity limited in extent is an almost certain sign of organic disease. General stiffness of the spine may be associated with conditions which cannot fairly be called disease, and which are of moment only by reason of the inconvenience or discomfort to which they give rise; the rigidity in advanced scoliosis may be given as an example. You see, therefore, that the whole subject of spinal rigidity is a little complicated, and it is also important, as very difficult questions may arise in connection with it. For an example let us take the following instance:—Not very long ago I was consulted by the parents of a boy about fourteen years of age who had a stiff spine; it was apparently stiff from

Digitized by Google

the sacrum to the cervical region. Upon testing him very carefully it was possible to detect now and again a sort of stammering in the rigid muscles about the spine. The ordinary tests did not show this, but on keeping the patient *for some minutes* in the bending position the rigid muscles began to stammer and then to give way to some extent, a thing which they never do under similar circumstances if there is organic disease, *e. g.* tuberculous disease. I therefore formed a primary impression that the trouble was psychic and not organic. I consequently advised massage and exercises, feeling pretty sure that the case was one of hysteria or neuro-mimesis; in other words, that the patient was afflicted with nervous mimicry of disease. This view caused some surprise and, indeed, indignation on the part of the parents, who had been assured by another surgeon that the boy was the subject of tuberculous disease of the spine, rest and spinal fixation for at least a year being advised, a very guarded prognosis at the same time being given. This I was only told after I had stated my opinion. I saw no reason to alter my views, and induced the parents to adopt the line of treatment I advised. In three months the boy went to a public school, and is now a soldier. I subsequently learnt that a relation with whom the boy had spent a good deal of time was undergoing treatment for tuberculous disease of the spine, which was being kept stiff by the use of a leather jacket. This fact led me to be more sure than ever that there was no disease in this boy's case, whose stiffness was due to his imitation of the rigidity in the other boy. The patient had, in fact, no disease of the spine at all; his stiff back was due to imitation of the rigidity from which his relation suffered. That is an excellent example to show how very necessary it is to keep on the alert for small symptoms, or rather very small defects in a series of symptoms, in the investigation of disease. If I had not, for example, in the course of my examination of this boy, kept him in the constrained position of flexion for a long time in order to fully estimate the degree in which his muscles were the cause of the rigidity, I should never have detected the stammering in the muscles when they became tired, and I should consequently have condemned the boy as a subject of tuberculous disease of the spine, because he had other signs which at first sight seemed to confirm the graver diagnosis. Jarring the shoulders, for instance, caused him to flinch; no doubt he had seen his relative flinch when tested in this way.

Rigidity of the spine, even if it be absolute and independent of muscular contraction, should not lead you to take it for granted that there is organic disease without inquiring further into the case. It is impossible to impress upon you too often or too strongly the great cardinal fact that disease is to be diagnosed by the observation of a series or collection of symptoms, and not by a single sign. In illustration, let us take the following case:—A medical man brought me his daughter, a girl about fourteen years of age, concerning whom he was in very great distress, as he had recently found that she was the subject of tuberculous disease of the spine of an advanced kind. He blamed himself greatly for not having noticed the condition until the lumbar spine and the last one or two dorsal vertebræ had become perfectly rigid. The girl had been going about, playing tennis and doing everything that a girl of her time of life should do, without complaining of any pain or showing any signs of ill-health. He had discovered the condition accidentally, his attention having been attracted to it by a certain stiffness of gait in his daughter which he had not noticed before. Upon examining her back he then found the rigidity concerning which he consulted me. I satisfied myself that the portions of the spine I have mentioned were absolutely stiff. There was no question of stammering muscles here. To any person not possessing a rather suspicious or inquiring turn of mind there would have been little to say about the case; it would perhaps have been said to be a quiet case of caries of the spine, either in a very latent or in a cured condition, because tuberculous disease of the spine does sometimes, as I hope you know, occur and run its course with an extraordinary absence of pain or other symptom of discomfort. Indeed, you sometimes find a patient with marked angular curvature of the spine, the result of tuberculous disease, in whom no pain whatever has been felt, and in whom no inconvenience has occurred, the condition having been found out accidentally. Here, then, was a case which under ordinary circumstances would have justified a somewhat serious opinion if judged merely from the amount of rigidity. But I have seen other cases like this, and I happen to know that there is a condition of rigidity of the lower part of the spine which is congenital; and

Digitized by Google

having this in my mind, I was struck with the fact, which I had not noticed before, that the father of this girl had a certain stiffness of gait, and I pointed out to him that so far as I could see there was very little difference between the gait of his daughter and his own; indeed, I ventured to go a point further and say that I should not be surprised to find that his back over the same area was stiff like hers. This suggestion was at first received with incredulity. I induced him, however, to let me make an examination, and I found that my impression was right. Going a point further still, we examined another daughter, who was rather younger, and we found that she also was quite stiff in the lumbar region of her vertebral column. This case affords an admirable example of absolute stiffness of the spine which would not have yielded under an anæsthetic, and which after all was no evidence of disease, being simply a congenital peculiarity—a very interesting condition, which is more common than is sometimes thought. Several other cases of this kind have come under my notice. I have seen, for example, congenital stiffness of the lower part of the cervical and upper dorsal region which had all the appearance of tuberculous disease with a slight angular curvature because a persistent stoop had led to an exaggerated projection of the vertebra prominens. The case occurred in a family of five children, three of whom showed the same condition. Congenital peculiarity is therefore a condition to bear in mind in estimating the importance of painless spinal rigidity.

I have said that rigidity of the spine is in itself no evidence of organic disease; on the other hand the absence of rigidity is no positive evidence of the absence of organic disease—an interesting fact. It is more common than is usually supposed for the spine to remain quite supple during the early stages of tuberculous disease, and in some cases during the formation of extensive psoas abscess. There was a very remarkable case of this kind in the Drummond Ward, in which a very large psoas abscess existed with no rigidity of the spine at all, and with no evidence of spinal disease beyond the psoas abscess. It was thought at first, therefore, that this abscess might not be connected with any disease of the spine, but that it was probably associated with some suppuration in tuberculous glands about the anterior aspect of the vertebral column; but as time went on and the abscess had to be treated by the radical method, there came out a piece of bone as large as a filbert; rigidity followed later—clear enough evidence that there had been all along organic disease. The explanation of such a case is probably not very difficult. The disease in the first instance was simply a tuberculous focus in the middle of the body of a vertebra, followed by quiet caries which, under ordinary circumstances, need not give rise to any rigidity, because there would not necessarily be any pain or irritation arising from it; and so until the disease extended beyond the original focus there was no rigidity, because no necessity for any protective muscular action arose. The case is an extremely good one to show that very distinct caries of the vertebral column, giving rise to a large psoas abscess, may develop without the least indication of any disease of the spine beyond the existence of the abscess; and fully demonstrates the truth of what I have just said, viz. that whilst rigidity of the spine is no positive evidence of disease, the absence of rigidity, on the other hand, does not exclude the possibility of tuberculous lesion,—in other words, organic disease may occur without rigidity, and rigidity may be independent of organic disease.

The causes of rigidity of the spine are numerous, and they vary to some extent at different periods of life. It may here perhaps be useful to remind you that any condition—such, for example, as injury, reflex irritation, or rheumatism—leading to contraction of the perispinal muscles will cause rigidity of the vertebral column so long as the muscular contraction lasts; the completeness of the spinal rigidity being in direct proportion to the degree of the muscular contraction. A small minority of the causes of rigidity operate equally at all times of life; injury may be given as an example. The majority, however, tend to confine themselves to certain periods. In *infancy and early childhood* the principal causes are injury; adenitis and glandular abscess around the column; worms, a cause very rarely mentioned; tuberculous disease of the column itself.

From the time of *puberty to the age of twenty-five* the causes are mainly injury; perispinal adenitis and glandular abscess; early cases of osteo-arthritis, which are not common; neuro-mimesis (neurotic or hysterical cases, as they are sometimes called); the congenital peculiarity to which I have

Digitized by Google

referred; advanced scoliosis; that rare condition called myositis ossificans; and finally, the most important, tubercle of the column. From *the age of twenty-five to fifty-five* a larger range of causes comes into play—namely, perispinal adenitis and glandular abscess, tubercle, malignant disease (most likely sarcoma), hydatid cysts, gumma, aneurysm, osteo-arthritis (spondylitis deformans), congenital peculiarity, injury, and myositis ossificans. In the *later periods of life, from fifty-five years onwards*, the causes become less numerous, being principally osteo-arthritis; spontaneous ankylosis, which is sometimes liable to occur in the joints of old people; the ordinary rigidity of old age, *i. e.* the loss of suppleness which comes upon most people at a late time of life; carcinoma, practically always "secondary" in character; congenital peculiarity; injury, of course; and rarely senile tubercle. You will note that I include the congenital cases only in those of the second period. The stiffness due to congenital peculiarity may be regarded as non-existent in infants, because, *in the absence of muscle rigidity or extensive tuberculous disease*, it is quite impossible to detect in an infant with any certainty whether the joints are stiff or whether they are not; because at that period, ossification not having taken place, the whole column is flexible, apart from any question of joints. Hence, on applying the lifting test which I described, the elasticity in the ossifying parts would convey a sense of bending to the hand of the investigator. In every other condition the lifting test is absolutely infallible in the youngest of subjects. In old age the tendency to spinal rigidity is too common from other causes to make the congenital condition of any clinical importance. There are other points relating to spinal rigidity which are of interest, but time does not allow of my dealing with them now. The matters of which I have spoken are, however, of practical utility, and are worthy of your attention.

Topical treatment of chronic pharyngitis:

℞ Iodi	. . .	gr. vj.
Pot. Iod.	. . .	gr. xij.
Ol. Menth. pip.	. .	♏ viij.
Glycerini, ad	. .	ʒ j.

To be applied by a camel's-hair brush.

A CLINICAL LECTURE
ON
SOME COMMON NERVE INJURIES, THEIR EXAMINATION AND DIAGNOSIS.

Delivered at the London Hospital.

By JAMES SHERREN, F.R.C.S.,
Assistant Surgeon to the Hospital; Surgeon to the Poplar Hospital for Accidents.

Gentlemen,—I want to draw your attention to-day to a few of the more common nerve injuries, their examination and diagnosis. It is my intention to keep strictly to the clinical side of the subject, and to leave untouched the scientific questions of regeneration and of the distribution of the various forms of sensation.

The conduction of impulses through nerves may be impaired or abolished by—

Anatomical division of the nerve, the result of a wound or rupture of the nerve; or

As the result of pressure, momentarily applied or long continued, resulting in a contusion, or in physiological division of the nerve.

I am using the term contusion to denote impaired conduction through a nerve, the result of subcutaneous injury, and the term physiological division to denote that conduction is entirely abolished without there being any division of the nerve-sheath.

The theoretical symptoms of anatomical division of a nerve—and by this I mean division of the anatomical nerve, axis-cylinders, and sheath—are well known to you all. They are loss of conduction of motor or sensory impulses, or both, according as the nerve is motor, sensory, or mixed, followed in a varying time by vaso-motor and trophic changes (I am using this word without committing myself to any theory as to the existence or non-existence of definite trophic nerves). Such are changes in the skin, nails, hairs, circulation, and muscles.

The symptoms caused by momentary pressure—for example, a blow or crush—vary from tingling in the area of distribution of a sensory nerve, or weakness of muscle in motor or mixed nerves, to signs of complete division; and this complete division may be anatomical, *i. e.* rupture, or may be physiological division. I want to impress upon

Digitized by Google

you that there is no method by which you can diagnose between anatomical and physiological division of a nerve the result of subcutaneous injuries, although the importance of having such a sign is obvious, as in the former case immediate operation would be carried out.

The two varieties may be combined, *e. g.* in a wound of nerve, where the nerve has been partly severed, and the conducting power through the unsevered part of the nerve interfered with as the result of physiological division; but here the diagnosis is of no practical importance, as it is necessary to expose the nerve.

The pressure may be longer continued and intermittent, as in crutch palsy; or gradual, the result of involvement of the nerve in callus or in new growth of any kind; and may result in signs of physiological division, but more often the signs are only those of contusion.

It is necessary to follow out some plan in examining a case of suspected nerve injury, or points are left out which are essential to diagnosis. A useful plan of examination is under the following three headings:

1. General Inspection of the Part injured: (*a*) position of injured part or limb; (*b*) position of wounds, scars, etc.; (*c*) condition of skin, nails, hairs.

2. Examination of Muscles: (*a*) inspection—for wasting; (*b*) action—for paralysis; (*c*) reaction to electrical stimulation.

3. Examination of Sensation: (*a*) to light touch; (*b*) to prick.

First we will consider the examination of a case of old nerve injury, such as one of those here to-day, or met with in the out-patient department or the examination room.

1. It is often possible to make the diagnosis that there has been a nerve injury by simply looking at the part. The position taken up is in some cases typical, *e. g.* in ulnar or musculo-spiral nerves. There may be changes in condition of skin, circulatory or inflammatory, with alteration in texture of the nails and growth of the hair, to tell you that there has been interference with the nerve-supply of the part.

2. By the wasting of muscles it is seen that a certain group is affected by the injury, and the knowledge is completed by seeing what muscles are paralysed, and what the electrical reactions of these muscles are. This electrical examination of muscles is exceedingly important, for it enables us to tell the degree of the nerve injury.

In testing, the faradic and galvanic currents are used, and the muscle may show changes in faradic reaction, galvanic reaction, or both. As you know from your physiology, the faradic current is a stimulus to nerve-fibres, the galvanic to muscle-fibres. The form of battery used for testing is the combined form such as I have here, where from a single pair of terminals all the testing may be done, instead of having to change the wires from faradic to galvanic batteries and then change the wire to alter the poles when using the galvanic current as a test. In testing, one precaution is necessary if you wish to get good results—the solution used to moisten the electrodes and the skin must be saline solution, and the part should be well soaked in it first. The faradic current is first applied; if the muscles react to this there is no need to go further, for we know that there is conduction through the nerve supplying the muscles we are testing. If we do not obtain contraction to the faradic current we know that the nerve is either anatomically or physiologically divided.

The galvanic current stimulates muscle and tells us the condition of the muscle itself. Muscle reacts to galvanism as long as there is any contractile substance left. In testing with galvanism start with the negative pole first—that is, have your testing electrode attached to the screw marked —, —then see what strength of current is necessary to get a contraction, note the kind of contraction obtained, and then switch over so as to reverse the poles and see if more or less current is necessary to make the muscle react to this pole.

Reaction of degeneration may be present, and briefly put it is this:

1. Loss of reaction to the faradic current.
2. Alteration of contraction to the galvanic.
   (*a*) Alteration in the character of the contraction.
   (*b*) Polar reversal.

Of the two the former is the more important.

3. I have noticed that many men have considerable difficulty over this, in knowing what tests to use, how to use them, and what conclusions to draw from their results. Two sensory tests are necessary —one for light touch and one for prick.

The best means of testing loss of light touch for

Digitized by Google

clinical purposes is to use a wisp of cotton wool. Go carefully over the part, telling the patient to shut his eyes and to let you know as soon as he feels the lightest touch; then mark out with blue pencil each time he feels it, and in this way you soon get the area of loss marked out. In testing for prick go over the area in the same way with a sharp pin and mark out its boundaries.

This area of loss of prick is never so extensive in injuries of peripheral nerves as is the area of loss for light touch. I wish to impress this upon your minds, for the absence of knowledge on this point has led to many wrong diagnoses.

made in the same three groups. When there is a wound:

1. Position of wound, whether over important nerve-trunks, etc. The position of the limb will give you very little information as a rule, because these cases are usually complicated with divided tendons. There are no alterations of course in skin, etc.

2. Muscular examination may reveal loss of power in certain muscles not directly affected by the wound; this, of course, will point to a nerve injury. The electrical reactions at first show no change.

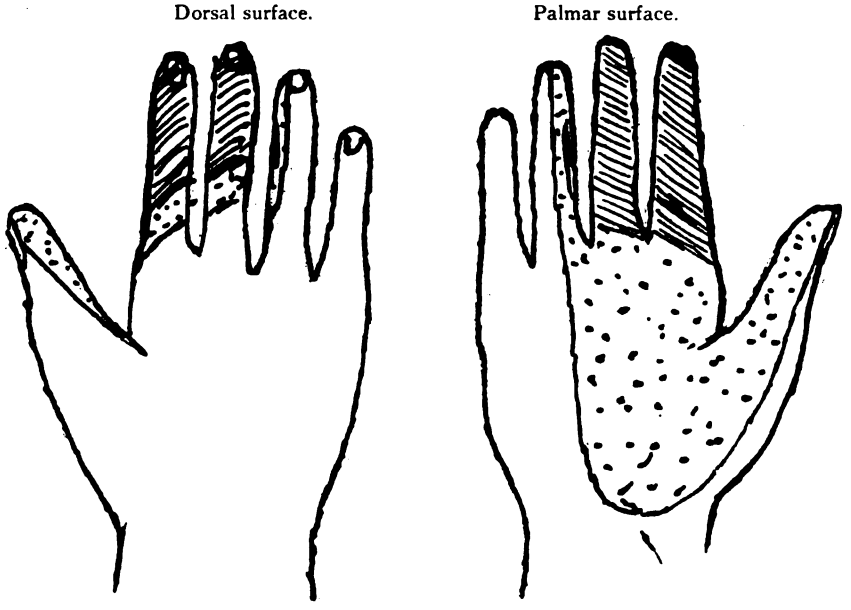


FIG. 1.—Anæsthesia after division of median nerve. Shaded area—loss to prick and light touch. Dotted area—loss of light touch only.

By the area of loss for light touch you are able to mark out the *extent* of the nerve injury, *i. e.* the nerve or nerves involved, your knowledge of anatomy telling you what they are. By means of the area of loss for prick you tell the *degree* of that injury, whether only partial or complete division of the nerve. These two are the only sensory tests necessary.

Heat and cold as tests are difficult to apply satisfactorily, and no further information can be obtained from them than given by light touch and prick. They may be neglected as clinical tests, though of course they are of scientific interest.

In cases of recent nerve injuries examination is

3. We have to rely for our diagnosis in these cases chiefly on changes in sensation. It should always be the rule to examine the nervous condition of parts below wounds, for it has happened many times that tendon suture has been performed and later the nerve injury discovered, thereby putting the patient to the inconvenience of two operations, as well as the difference of prognosis between primary and secondary suture.

In subcutaneous injuries, particularly in fractures likely to be complicated with nerve injuries, it is important that the diagnosis should be made before the limb is put up, or it may happen that it is first noticed when the splints are taken off, and attributed to pressure by splint or callus. The signs which you

Digitized by Google

have to rely on are as above, but of course here, where you have no wound, the position of the limb and action of muscles are more important.

In diagnosis there are two factors: first, the recognition of a nerve injury and the particular nerve or nerves injured; secondly, the degree of that injury. Both are told by carrying out the complete examination, but I should like to draw your attention first to the second factor. The *degree* of involvement of the nerve is told by the area of loss of sensation and the electrical reaction of the muscles. If the loss for light touch and prick together or separately is markedly less than should be present in injury of that particular nerve, you know that the nerve is not divided. The electrical reactions give you often the most important clue. If the muscles react to faradism you know that the nerve supplying those muscles is not divided. In nerves which are chiefly muscular, as the musculo-spiral, this is the only means we have of saying whether that nerve is contused or divided. I should just like to remind you here that the electrical reactions of the muscles are at first practically normal, and only lose their faradic irritability about the fourth or fifth day after division of their nerve-supply.

There are two conditions following injury that are occasionally diagnosed as nerve injuries: one is hysterical anæsthesia, paralysis, or both, and the other is Volkmann's contracture.

Unfortunately, I have not an hysterical limb here to show you to-day. This form of functional disease may arise in direct relation to an injury of the part affected, and the patient complains of loss of power, of numbness, or both.

In examining such a patient, you see that the limb is hanging helpless, and there may be circulatory changes. If the condition has persisted for any length of time there will be wasting of muscles.

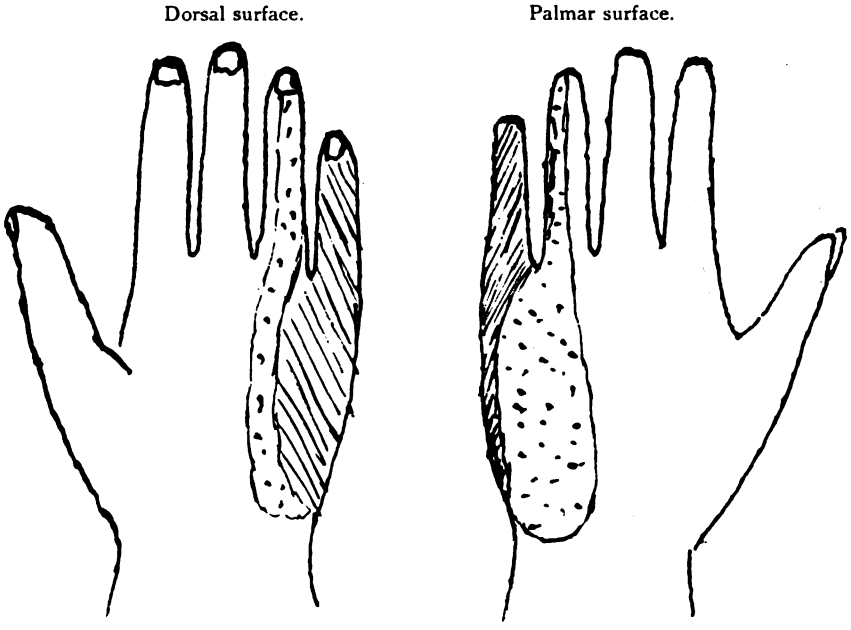


FIG. 2.—After complete division of the ulnar nerve. Shaded area—loss of prick and light touch. Dotted area—loss of light touch only.

In continuing the examination of the muscles, you find that those paralysed do not correspond to any known combination of peripheral nerves, and that the electrical reactions of those muscles are normal.

The loss of sensation usually extends up to a joint, and its upper limit encircles the limb as a ring. Up to this line light touch and prick are lost, and you know that there is no nerve injury that will produce this loss of sensation.

Volkmann's contracture is a contracture of muscles—usually of the flexors of the forearm,

Digitized by Google

following an injury, usually the application of splints to the forearm.

I have here a case which is at present under the care of Mr. Moullin, and which he has kindly transferred to me for use this afternoon and treatment later.

On Easter Saturday she sustained a fracture of the forearm. This was treated by splints with the result you all can see.

She shows very beautifully the phenomenon that is characteristic of Volkmann's contracture. With the wrist extended, the fingers are flexed at the interphalangeal joints, and cannot be extended, actively or passively. With the wrist flexed the movement of extension can be carried out. You see that she can flex her fingers herself, showing you that the muscles are not paralysed.

In Volkmann's contracture there is no change in sensation, unless, as in this case, nerves be injured by pressure at the same time, but I wish to impress on you that the nerve injury has nothing to do with the contracture. The electrical reaction of these muscles is normal, as is always the case. To be aware of this condition, and to have seen a case is sufficient to keep you from a wrong diagnosis.

Now I want to go over with you a few points in connection with some common injuries and to show you a few cases. I have here a typical case of division of the median nerve. This nerve is usually injured in cuts just above the wrist, caused in the majority of cases by putting the hand through a glass window. There is just one point in the anatomy of the nerve that I wish to draw your attention to. This nerve occupies a very superficial position just above the wrist, lying between the tendons of flexor carpi radialis and the outermost of flexor sublimis, and here it is sometimes injured without injury to any of the surrounding tendons. For that reason the injury has sometimes escaped notice, as the rule of examination for loss of sensation below wounds was not followed, and tendon injury did not necessitate exploration of the wound. There is nothing much that is typical in the position assumed by the hand; usually the thumb is kept close to the index finger, the so-called "ape hand." In muscles, there is wasting of the outer part of the thenar eminence occupied by the abductor and opponens pollicis muscles, with loss of abduction and opposition of the thumb.

Abduction and adduction of the thumb are

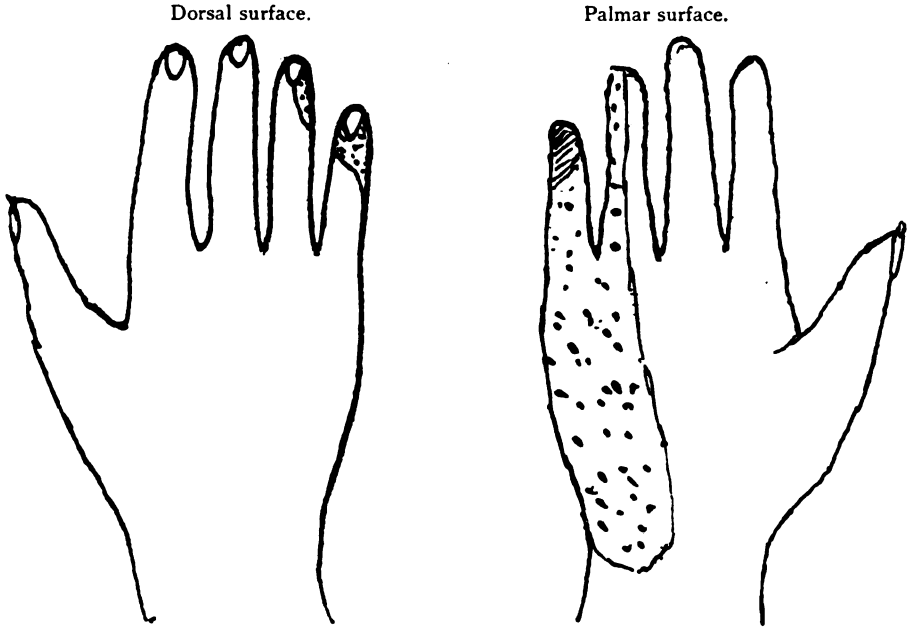


FIG. 3.—After division of palmar branch of ulnar nerve.

Digitized by Google

made in a plane at right angles to the palm of the hand and not in a plane parallel to it, as are abduction and adduction of the fingers. This is owing to the different plane occupied by the metacarpal bone of the thumb.

Opposition is swinging the thumb round until its anterior surface faces the palm.

There is loss of the sensation of light touch on the palm of the hand and the palmar surface of the fingers corresponding to that given you in anatomical text-books, the thumb and two and a half fingers with corresponding part of the palm.

The loss of prick is usually limited to the index and middle fingers, though it may occasionally extend into the palm or involve the thumb, but is never less than that seen on this diagram (Fig. 1). On the dorsum there is loss of light touch and prick over the first and second phalanges of index and middle fingers, the area for light touch being only a little larger than that for prick. There is no loss of sensation over the dorsum of the thumb. This dorsal area of anæsthesia, as you see, differs somewhat from that given in anatomical text-books.

The ulnar nerve is usually injured in the same way as the median in wounds about the wrist, but rarely without injury to tendons. It may also be injured by pressure in fractures about the internal condyle of the humerus, in blows in that situation, or by pressure from splints.

There is one point about the anatomy of this nerve also that I wish to remind you of. The nerve divides about two inches above the line of the wrist into palmar and dorsal branches; consequently in wounds just above the wrist the dorsal branch escapes. Forgetfulness of this point has led to the diagnosis being missed and the wound closed, the patient coming under observation later because of wasting of the muscles of the hand. It is only necessary to mention this to prevent any of you making a similar mistake.

In division of the ulnar nerve the position taken up by the fingers is typical, the so-called "claw hand" with abduction of the little finger. Muscular wasting of the hypothenar, inner part of thenar eminences, and interossei muscles is marked. All interosseous movement is lost, and there is loss of adduction of the thumb and abduction of the little finger.

Anæsthesia to light touch, if the whole nerve be divided, corresponds to that given in your anatomy books. If only the palmar branch, then on the palmar surface it is the same; but on the dorsum there is loss over the terminal phalanx of the little finger only. The boundary of the loss for light touch is along the ulnar border of the hand, as you see in the diagram. Loss of prick when the whole nerve is divided takes in the whole little finger and a strip on the ulnar border of the hand, both on the palmar and dorsal surfaces. If the dorsal branch be intact the loss of prick varies, but as a rule is limited to the tip of the little finger (Figs. 2 and 3).

I cannot leave the subject of the ulnar nerve without impressing upon you the importance of this palmar branch. As you know, it carries with it the nerve-supply for the muscles of the hand. If, after a wound involving this branch only, you test as I have seen so many do—take up a pin and stick it in the palm of the hand,—you will miss this; for, as you see, the loss of prick is limited to the tip of the finger, and you must make it a rule to examine systematically, starting with light touch.

An injury to the ulnar nerve is, as you see, a much more serious accident than injury to the median, and means, if recovery does not take place, a useless hand.

When the median and ulnar nerves are divided together, the position of the hand resembles that seen after division of ulnar. There is marked wasting of all the intrinsic muscles of the hand, and loss of movement of these muscles. The anæsthesia, as you see from this diagram, is for light touch, the median and ulnar areas together. The loss for prick, as you see, is a little more extensive, extending further in on the ulnar side of the palm.

The musculo-spiral nerve is very rarely injured by wounds, but is very frequently injured as the result of pressure in fractures of the humerus, at the time of the fracture, or by involvement in callus later; or in cases of crutch pressure, as in this case; or in the so-called "Saturday night palsies." The lesion is easily recognised—the drop wrist with loss of power in the extensors of fingers and wrist, and in the supinator longus. As a rule there is little or no loss of sensation.

I am showing you this case because it is a

Digitized by Google

typical musculo-spiral injury, and because it shows well the results of a contused nerve—a nerve whose conduction has been impaired as the result of pressure. The boy first came under my observation in April, and his left arm was typical of complete musculo-spiral injury, and his right arm was in the condition that the left is in now,—that is, partial recovery of the extensors of the fingers and of the supinator longus. The right arm has now completely recovered.

On my first examination none of the extensor muscles were acting, but it was only on testing those muscles electrically that I was able to tell whether the nerve was physiologically divided or only contused. The muscles reacted to faradism, and I was able to say that the nerve was only contused and that the arm would recover speedily, and it is doing so. This will again impress upon you the necessity of electrical testing in diagnosing the degree of the injury.

I am showing you this case of external popliteal injury as a typical case of this nerve injury and as an illustration of pressure resulting in complete physiological division. He was in this hospital under the care of Mr. Mansell Moullin, by whose kindness I am permitted to use him to-day. The injury took place nine months ago, and was due to the fall of a bale of wool on the outer side of the leg. The external popliteal nerve is in an exposed condition here, and is most often injured as the result of pressure.

The most common pressure is the use of Clover's crutch in operations. As a rule it causes only slight anæsthesia and a little weakness of muscles, which passes off in a day or two, but it may lead to a condition which takes months to clear up. The nerve may also be injured by involvement in callus in fractures in the region of the head of the fibula. It may also be injured during tenotomy of the biceps flexor cruris.

The foot in this injury, as you see, is in the position of talipes equino varus. There is wasting of the anterior and external groups of muscles, with loss of extension of the toes, dorsi-flexion and eversion of the foot. These muscles give the reaction of degeneration when, as in this case, the nerve is divided.

The anæsthesia to light touch involves an area such as I have marked out, and I want you to notice that sensation is retained over the terminal phalanges of the outer four toes. This loss of light touch practically corresponds to that given in anatomy books. Prick is lost over an area a little less in all directions than the area of loss of light touch. If the nerve be injured above its lateral cutaneous branch the loss extends higher up the leg.

I want you to carry away from this case the knowledge that a blow over a nerve, not causing rupture, may give all the signs of complete division, and take as long to recover as if the nerve had been divided and primary suture done, and may not recover without operation.

LARGE MONONUCLEAR LEUCOCYTOSIS IN MALARIA, BLACKWATER FEVER, AND LYMPHADENOMA.

By FRANK BUSHNELL, M.D.,

Hon. Pathologist, South Devon and East Cornwall Hospital, Plymouth; Lecturer on Bacteriology, Plymouth Education Authority, School of Science and Technology.

THE diagnostic value and the meaning of large mononuclear leucocytosis in disease is of much interest. On this account I am induced to place on record cases of malaria, blackwater fever, and lymphadenoma in which it was present (see Table, Cases 1, 2, and 3).

To illustrate the subject further I have added blood counts of trypanosoma, sarcoma, etc., from other sources (see Table, Cases 4, 1*a*, 2*a*, 3*a*, 4*a*, and 5*a*).

The subdivision of non-granular cells is occasionally easy, at other times difficult or impossible, with ordinary clinical methods of blood examination.

For the consideration of the cases I append a *résumé* of recent views on this debatable point as not out of place. The varieties usually enumerated are the lymphocyte, the large lymphocyte, the large mononuclear leucocyte, and the transitional.

With Louis Jenner's staining mixture of eosin and methylene blue in methylic alcohol, the

Digitized by Google

lymphocyte is a small cell with very little cytoplasm and a deeply staining nucleus. I find that the morphological distinction between the large lymphocyte and the large mononuclear leucocyte is frequently impossible, but I have attempted to separate these cells because their source and function probably differs. I place under the heading "large mononuclear" all cells with rounded or indented nuclei which in size are equal to or larger than the polymorphonuclear "neutrophile." Their colour with eosin and methylene blue is reddish or purplish and pale,—in other words, the cytoplasm is feebly basic; it is abundant as a rule, and may or may not contain granules, and the nucleoplasm may be more intensely stained than the cytoplasm.

The large lymphocyte stains blue, and frequently its nucleus stains less deeply than its protoplasm, which may contain basic granules and be scarce in amount. The lymphocyte is the small cell with very little cytoplasm and a deeply staining nucleus.

The transitional cell resembles the large lymphocyte in shape and size, but its nucleus is indented markedly and its protoplasm is purplish.

Ehrlich asserts that the distinction between large mononuclear cells and large lymphocytes can always be maintained. Cabot and Da Costa (1) do not consider that any distinction can be drawn between them in specimens stained by routine methods. Louis Jenner (2) separates them and characterises the large mononuclear cell as having a cell-body larger and paler than the lymphocyte, and free from granularity. Captains L. Rogers, I.M.S., and T. H. Delaney, I.M.S. (3), class all single unlobed nucleated cells as large or larger than a polymorphonuclear as "large mononuclears," and transitional cells as "polynuclear." Melland states that the "large mononuclear" compared with the large lymphocyte possesses much more protoplasm, in which the nucleus is usually excentrically placed, whilst both the nucleus and the protoplasm stain more faintly, and the margin of the nucleus is ill-defined. Melland also includes with these all transitional cells which have all the same characters, except that the nucleus, instead of being round or oval, is more or less deeply indented; and he considers their classification in practice not difficult (4).

Boycott and Haldane include under "large hyaline" the "well-known cell of large size with a (usually) indented nucleus, which does not stain deeply, and fairly abundant cytoplasm containing abundant basophile granulations." The lymphocyte they define as small cells with very little cytoplasm and a deeply staining nucleus. Between these two well-marked classes are a considerable number of cells which in size and other characters lie in an "intermediate" position and are classed as "intermediate" cells (5).

I have placed under the heading "large lymphocytes" in all probability these "intermediate" cells and the "abnormal mononuclears" of Whyte and Daniels (staining deeply with Leishman's stain) (6). In tint and size and contour of cell and nucleus, in relative proportions of the former to the latter, in the situation of the nucleus, and in the staining (ordinary) properties of cyto- and nucleoplasm, I believe that, at times, all grades are exhibited between the large lymphocyte and the large mononuclear cell.

In Case 1, malaria intermittent, the high percentage of 59·7 per cent. of lymphocytes was found, consisting of 32·5 per cent. of large mononuclear cells (3342 in 1 cm.), 12·0 per cent. of large lymphocytes, and 15·2 per cent. lymphocytes. This marked relative lymphocytosis occurred at the expense of the polymorphonuclear "neutrophils" (35·6 per cent.), and was present at three examinations: February 2nd, 1903; February 9th, 1903; and March 3rd, 1903. On February 21st the polymorphonuclears rose to 46·6 per cent. and an eosinophilia of 19·3 per cent. showed itself, the patient having an inguinal bubo, thought to be secondary to irritation from a "jigger" in the foot. The lymphocytes fell to 32·7 per cent. at this examination, with 12·7 per cent. large mononuclears, 4·4 per cent. large lymphocytes, and 15·6 per cent. lymphocytes.

Case 2 (blackwater fever, with few crescentic or ovoid bodies in blood).—This count was taken March 4th, 1903, at the end of a severe attack of blackwater fever, the patient being very anæmic and cachectic; the evidence of past malignant malarial infection being a few crescentic bodies in the blood. The leucocytes numbered 25,591 per cm.; and as many as 30,000 per cm. is given in certain pernicious malarias (7), and in certain stages of the paroxysm (8) there was an absolute increase of large lymphocytes and large mononuclear cells to

Digitized by Google

21·6 per cent. on one occasion and 26·5 per cent. on another. Of these 9·9 per cent. (2533 per cm.) and 11·1 per cent. were large mononuclear cells, and 11·7 per cent. and 15·4 per cent. were large lymphocytes.

Incidentally it is of interest to note that the history of this case confirms the fact that blackwater fever and malaria are associated (Davidson, Cross, Prout, Plehn, and Manson) (9).

The patient had been on the west coast of Africa since the beginning of 1902. He had a "short attack of malaria" in February, 1902, and an attack of "fever" in February, 1903. He had taken in all a few hundred grains of quinine only.

On February 20th, on his homeward voyage, blackwater fever developed, with urine like treacle and becoming solid on boiling. When the urine was examined in Plymouth there was "*débris*" at first, but the spectroscope did not reveal the blood. Crystals resembling leucin and tyrosin were noticed, and also oval bodies. On March 17th the patient had a sharp attack of "malaria." He was not taking quinine at the time, and on administration of quinine in doses of five grammes of the sulphate no hæmoglobinuria occurred.

The lymphocytes in malaria are well known to attain to 60 per cent. or more from the normal 20 per cent. to 30 per cent. Christophers and Stephens have found an excessive percentage of large mononuclear cells (10), as also Türck, Rogers and Daniels, and Delaney. Melland considers 10 per cent. to 12 per cent. of large mononuclear cells as strong presumptive evidence of malaria. L. Rogers would fix 12 per cent. as a limit above which a large "mononuclear" percentage is diagnostic of malaria.

It is to be remembered that most authors refer to a relative increase of lymphocytes or large mononuclears in both malaria and blackwater fever (11). Stephens states that this relative increase is least marked during the pyrexia and greatest during the apyrexia, and may persist in some cases with leucocytosis as well as leucopenia.

Cases 3 and 5 (lymphadenoma).—In Case 3 there was a distinct increase in the percentage of large lymphocytes *plus* large mononuclear cells to 25·3 per cent., consisting of 17·3 per cent. of large mononuclears and 8 per cent. large lymphocytes (7016 per cm. and 2782 per cm.). In Case 5 the large lymphocytes rose to 16·8 per cent., and the total lymphocytes to 52·2 per cent., without any leucocytosis.

This occurred with a leucocytosis of 34,781 per cm. without any secondary infection being noted, but as a rule no leucocytosis occurs in lymphadenoma, but a relative lymphocytosis of either type of cell with leucopenia. Da Costa has observed 70 per cent. to 80 per cent. of lymphocytes, chiefly large, in one case.

It will be seen from reference to the Appendix that relative increase of large lymphocytes occurs in trypanosomiasis, blackwater fever, lymphadenoma, and sarcoma. Lymphocytosis is also known to be a frequent accompaniment of tuberculosis, and in splenic and lymphatic diseases, terminal or acute infections, and after hæmorrhage, in severe anæmias, in cachexia, in lactation, in syphilis, in Graves' disease, and after the injection of tuberculin or the administration of thyroid extract. In infants and young children it is physiological (60 per cent. is within the normal limit), and often pathological.

In the light of the many sources of error that present themselves in attempting to subdivide the non-granular cells when stained by the ordinary methods, and recognising the various diseases in which lymphocytosis exists, the conclusion seems obvious that the diagnosis of any one disease from this single feature must be most guarded and provisional—if, indeed, it is possible. On the other hand, such a relative excess of lymphocytes as 59·7 per cent. is certainly suggestive of malaria, though not diagnostic. Cases 1, 3, 4, and 5 were four out of the last thirty-four examinations made in the Clinical Laboratory of the South Devon and East Cornwall Hospital, and show lymphocytosis.

Lymphocytosis may be due either to changes in the distribution of the cells through the circulatory system, or to their increased production and output by the lymphatic tissues. Ehrlich regards the large lymphocytes as the mothers of the small ones, and the large mononuclear cells and transitionals as myelogenous in origin. Uskow believes that the lymphocyte is the youngest form of cell, and is derived from lymph-glands, lymphocytic bone-marrow, and spleen, and that this enlarges to the large lymphocyte, large mononuclear, polymorpho-

Digitized by Google

nuclear, etc., partly in the blood-stream and partly in the tissues.

The meaning of lymphocytosis is obscure. Ehrlish attributes it to "local irritation of certain areas of lymphatic glands which produces an increased circulatory activity in these situations, in consequence of which large numbers of lymph elements are swept mechanically from the lymphatics, and enter the general circulation. Ehrlich does not regard the change as an expression of an active chemotactic reaction, to which the lymphocytes are insensible" (12).

Da Costa suggests that the lymphocytosis which accompanies leucopenia may be traced to negative chemotaxis, which diminishes the number of polynuclear neutrophiles and thus brings about a relative increase in the lymphatic elements, upon which the repellant action is not exerted (13).

That large mononuclear cells do appear in response to positive chemotaxis has been proved by Metchnikoff and others in the guinea-pig. Metchnikoff states that the injection of *cells* into the peritoneal cavity of the guinea-pig is followed by an immigration of phagocytes (macrophages) which take up the alien cells. Having done this they wander back into various organs, meanwhile digesting the ingested cells by means of a hypothetical cytolytic ferment "cytase" (macrocytase) (complement).

Microphages (polymorphonuclears), on the other hand, appear in response to bacterial infections, and manufacture "microcytase."

The cytase is not free in the blood, but is secreted by the leucocytes, since no hæmolysis occurs on subcutaneous injection of xanthocytes (14).

Gengou has shown that exudates of macrophages had hæmolytic, but not bactericidal properties; microphages possessed bactericidal, but no hæmolytic powers (15).

According to Metchnikoff, it is the large mononuclear leucocytes (the only form of leucocytes in the alligator) which produce antitoxin, and he supports this view by experiments, showing that the poisons can be found fixed in the macrophages of warm-blooded animals while the surrounding fluid is free. The probability is that leucocytes play a considerable but not exclusive part in the formation of antitoxin, and that the macrophages have a preference, according to Metchnikoff, for animal cells, as erythrocytes; for the bacteria of chronic disease, *e. g.* tubercle and leprosy; and for the malarial and other analogous parasites (16).

Clinically, I believe that I have observed a relation between diseases of the lymphatic glands in infants and young children, with or without lymphocytosis, and a diet of artificial (patent) foods. It might be considered in these cases that the lymphatic glands and lymphocytes suffer a perversion of their normal habits and functions by a vicious departure from the natural human ingesta (17).

Cases 1, 2, 3, 4 and 5 were under the care of the staff of the South Devon and East Cornwall Hospital, to whom I am much indebted for the notes.

BIBLIOGRAPHY.

(1) Da Costa, 'Clinical Hæmatology,' p. 161.

(2) Allchin, 'Manual of Medicine,' vol. ii, pp. 290, 291.

(3) Captain T. H. Delaney, I.M.S., 'Brit. Med. Journ.,' March 28th, 1903, p. 725.

(4) C. H. Melland, 'Brit. Med. Journ.,' September 27th, 1902, p. 965.

(5) A. E. Boycott and J. S. Haldane, 'The Journal of Hygiene,' vol. iii, No. 1, January 1st, 1903, p. 115.

(6) Duncan Whyte and Daniels, 'Brit. Med. Journ.,' May 30th, 1903, p. 1251.

(7) P. Manson, 'Tropical Diseases,' pp. 98, 99. Ed. 1900.

(8) L. Rogers, 'Brit. Med. Journ.,' April 5th, 1902.

(9) Da Costa, 'Clinical Hæmatology,' p. 382. Allbutt, 'System of Medicine,' vol. ii, pp. 745, 746.

(10) Christophers and Stephens, 'Royal Soc. Malaria Committee Report,' 5th series, 1901.

(11) Stephens, 'Lancet,' 1901, vol. i, p. 848.

(12) Da Costa, 'Clinical Hæmatology,' p. 197.

(13) Da Costa, ibid., p. 197.

(14) A. S. F. Grünbaum, 'The Lancet,' March 21st, 28th, and April 4th, 1903.

(15) A. S. F. Grünbaum, ibid., March 21st, 28th, and April 4th, 1903.

(16) A. S. F. Grünbaum, ibid., March 21st, 28th, and April 4th, 1903.

(17) F. Bushnell, 'Brit. Med. Journ.,' September 12th, 1903, p. 585.

Digitized by Google

No.	Name, age, and sex.	Disease, etc.	Red cells per c.mm.	Hæmoglobin per cent.	Colour index.	Total leucocytes per c.mm.	Large mononuclear cells. Large lymphocytes.	Lymphocytes.	Transitional.	Polymorphonuclear neutrophile.	Eosinophile.	Mast-cells.	White to red.	Total of large mononuclears per c.mm.	Date.
1	S. T., 28, male	Malaria (æstivo-autumnal); examination made at end of paroxysm, temperature being 99·2°; no quinine given; "crescentic bodies" and "rings" abundant	2,480,000	50 Haldane's modified Gowers	1·0	10,284	32·5, 12·0 = 44·5	15·2	0·9	35·6	3 5	0	1 to 240	3342	Feb. 2nd, 1903.
		Second examination	2,560,000	50	1·0	12,891	30·7, 14·2 = 44·9	17·7	0·9	27·9	8·2	0	—	—	Feb. 9th, 1903.
		Third examination (bubo and jigger in foot)	2,800,000	75	1·2	14,673	12·7, 4·4 = 17·1	15·6	1·2	46·6	19·3	0	—	—	Feb. 21st, 1903; bubo.
		Fourth examination, apyrexia	—	—	—	—	32·9, 15·6 = 48·5	17·5	1·8	27·9	13·5	·005	—	—	Mar. 3rd, 1903.
2	A. B., 35, male	Blackwater fever and cachexia; a few malarial crescents in blood; blood examination made at end of the hæmoglobinuria.	2,420,000	30	0·6	25,591	9·9, 11·7 = 21·6	9·9	1·0	64·6	2·7	0	1 to 94	2533	Mar. 4th, 1903.
			—	—	—	—	11·1, 15·4 = 26·5	20·8	0·5	49·5	2·4	0.			
3	E. K., 18, female	Lymphadenoma (no signs of secondary infection)	6,000,000	75	0·7	34,781	17·3, 8·0 = 25·3	8·5	2·0	62·2	1·5	0	1 to 170.		
4	W.N., 17, male	Sarcoma of scapula	—	—	—	—	12·5, 8·2 = 20·7	8·8	—	66·8	1·7	0.			
5	P., 11, male	Lymphadenoma (cervical glands on right side much enlarged)	5,100,000	76	0·75	12,000	16·8	35·4	1·1	38·5	8	1	1 to 425	—	350 cells counted Sept. 16th, 1903.
1*a*	E. M., 26, male	Malaria crescents in blood	3,240,000	—	—	4,600	17	28	—	53·0	2·0	0	1 to 704.		
2*a*	T. R., 25, male	Blackwater fever; temperature 99·6°	3,425,000	65 Von Fleischl	—	10,000	18	21	—	59	1	1	1 to 342	—	Mar. 9th, 1903.
			3,635,000	65	—	7,000	—	—	—	—	—	—	—	—	Mar. 23rd, 1903.
			3,00,0000	59	—	10,000	8	13	—	77	—	1	—	—	Mar. 24th, 1903.
			3,000,000	56	—	11,800	21	20	1	56	2	—	—	—	Mar. 25th, 1903.
3*a*	S., 40, female	Trypanosomiasis	3,880,000	—	--	8,600	29·7	16·0	—	51·5	1·0	0·25	1 to 451	—	Nov. to Dec., 1902.
			2,825,000	36	—	4,200	22·7	18·4	—	55·2	1·8	0·48	—	—	" " "
			—	—	—	—	19·6	24·0	—	53 7	2·5	0·15	—	—	Feb. to Mar., 1903.
4*a*	—	Lymphadenoma	3,853,700	58·5	0·78	10,480	13·3	15·1	—	69·3	1·8	—	—	—	
5*a*	—	Osteosarcoma of thigh	—	—	—	Great increase	6	19	—	74	1.				
6*a*	male	Normal average of cells per c.mm.	5,000,000	100	1·0	7,500	4 to 8	20 to 30	—	60 to 75	0·5 to 5	0·5.			
	female		4,500,000	80·90 Von Fleischl											
7*a*	—	—	—	—	—	5,000 to 10,000	200 to 800	1,000 to 3,000	—	3,000 to 7,500	25 to 500.	25.			

1, 2, 3, 4, and 5, Reports Clinical Laboratory, South Devon and East Cornwall Hospital, Plymouth. 1*a*, Captain T. H. Delany, M.B., I.M.S., 'Brit. Med. Journ.', March 28th, 1803. 2*a*, Drs. W. G. Ross and G. C. Low, 'Brit. Med. Journ.' 3*a*, Drs. Duncan, Whyte, Daniels, and Lowe, 'Brit. Med. Journ.,' May 30th, 1903. 4*a*, Dr. J. C. Da Costa (average of ten cases). 5*a*, Dr. R. C. Cabot (Clinical examination of the blood). 7*a*, Dr. J. C. Da Costa, total number of leucocytes normally found per c.mm. ('Clinical Hæmatology').

Digitized by Google

INTERNATIONAL CLINICS. Thirteenth series, vols. i and ii, 1903. (London: J. B. Lippincott Company.)

The first of these volumes opens with a valuable article on aneurysm of the descending aorta by Dr. Osler. Fourteen cases are reported in full, and the whole subject is discussed under the headings of etiology, symptoms, diagnosis, and treatment. "Fourteen cases with thirteen deaths is a comment upon treatment!" He adds, however, that in a majority of the cases the suffering was greatly relieved, and that this in such a hopeless disease is to fulfil the highest function of the physician. We note with interest that in none of the cases did Dr. Osler think it advisable to try the method of wiring, with electrolysis, although the question was considered. Big operations with big names are described by Messrs. Keen, Senn, and Tonnesco, but we regard a reiteration of their triumphs as more suited to operating surgeons than to the general practitioner. A very useful article on convulsions in young children is contributed by Dr. John Thomson, of Edinburgh, which will prove extremely helpful to all who meet with such cases. He places himself in the position of the family practitioner called suddenly to a baby in a fit, and more perturbed by the questions put to him by the anxious relatives than by the condition of the patient. Dr. Thomson attends to both the patient and the friends by answering fully the following questions:—What is to be done while the child is in the fit? Is the convulsion likely to be fatal? What is it caused by? Is it likely to be followed by others? Will the child's intellect or future health suffer? What is to be done to ward off further seizures? The editorial supply of clinical lectures seems to have run out, and a hundred pages of this volume are devoted to a retrospect of medicine and surgery in 1902. The letterpress is too brief to be of much value to any one, and the same subject is much better treated in other works; but the illustrations here are a very special feature, and form a most instructive picture gallery. The second volume opens with a symposium on the summer diarrhœas of children, contributed by Drs. Conn, Hand, Cotton, Westcott, Nicoll, and Marfan. Curiously enough, this season has been characterised in London by less summer diarrhœa than for many years. One factor in preventing this disorder is referred to by several of the writers—namely, the thorough flushing of the streets by the authorities in hot weather; and although our own authorities do not require special thanks in this connection, the torrential rains so frequently repeated have probably been the means of saving many lives from death from diarrhœa. The statements and views of the above-mentioned writers are very full and very true, but not strikingly new. We have read with most pleasure the article by Professor Marfan, of Paris, in which he gives his own treatment based on his own experience. As he has entirely given up the washing out of the stomach and the bowel, the use of alcohol, and the administration of such drugs as salol, bismuth, calomel, and opium in the acute stage, it will be seen that routine treatment has no special attraction for him. We turn with interest to another article on the etiology and treatment of a common cold by Dr. Haig, but only to read that a "cold is a gout of a respiratory mucous membrane." The mighty atom of uric acid is also at work even in this humble and much-neglected department of medicine! The "rest treatment" has been much written about and much employed since its introduction by Dr. Weir Mitchell, but we have read nothing better than a paper in this volume by Dr. Madison Taylor on the indications for its use and the methods by which it is to be carried out. As the details of the treatment in each individual case are of vital importance, we commend this paper to the study of all who are interested. There are several other articles in this excellent volume which will repay careful perusal.

THE INTERNAL SECRETIONS AND THE PRINCIPLES OF MEDICINE. By C. E. de M. SAJOUS, M.D. Vol. i. (Philadelphia: F. A. Davis Company, 1903.)

This will be, when completed, a very large work. Now only the first volume lies before us, but it extends to 800 pages of closely printed matter. The feeling uppermost in our mind after reading it is that its very length is its fault. The author's ability to write easily is his misfortune, for it prevents that compression which is necessary to make the book generally useful; and further, in his desire to be full, the author gives the views of various authorities without perhaps sufficient indication of their respective value. But having said this, we have said all that can be urged against the book. Much can be said for it, because the author has most laboriously compiled a large amount of information about some of the most important questions in medicine, such as the physiology and pathology of the supra-renal glands and pituitary body, and the relation of internal secretion to immunity. Those who are specially working at these subjects will find the text of the book and its foot-notes of references very useful, and we believe it will be largely consulted by original workers. To such we recommend the work. It is clearly impossible for us to criticise such a mass of information in any detail.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 577. WEDNESDAY, NOVEMBER 18, 1903. Vol. XXIII. No. 5.

CONTENTS.

PAGE

*A Demonstration of Cases at St. George's Hospital. By A. MARMADUKE SHEILD, M.B., F.R.C.S. 65

*Two Clinical Lectures on Aneurysm of the Abdominal Aorta. By J. H. BRYANT, M.D., F.R.C.P. LECTURE I ... 71

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½, *Bartholomew Close, London, E.C.*

A DEMONSTRATION OF CASES AT ST. GEORGE'S HOSPITAL.

By A. MARMADUKE SHEILD, M.B., F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—Here is a patient with fractured ribs. Now fracture of the ribs is one of the commonest accidents which you may meet with in practice, and therefore it is very important that you should know all about it. The importance of fractured ribs depends mainly upon the structures which may be injured inside the chest, or the abdomen, by the broken bone. The structures frequently injured in the chest are the lung and pleura. The structures which are commonly injured in the abdominal cavity are the spleen or the liver. It is particularly important to remember that in a bad fracture of the lower ribs, especially from great violence, such as when a man gets caught between the buffers of an engine and carriage, there may be serious injury of the abdominal viscera. The liver is very readily torn as a result of fracture of the lower ribs, and it is a serious complication too often overlooked. A drunken heavy person may fall downstairs, his ribs may be broken and penetrate the liver and may cause extravasation of bile and blood into the peritoneal cavity, and all this may be quite unsuspected. When students write out the complications of fractured ribs they almost always omit to mention the possibility of damage to the abdominal contents. The diagnosis of fracture of the ribs is often inferential,—that is to say, you cannot be always quite certain of it. I want especially to draw your attention to the right method of examining a patient in whom you suspect this accident. The proper way to carry out such an examination is as follows:—Let the patient lie down on his back, place the arms by the sides, and then bend up his lower limbs so as to relax the muscles as much as possible. Then you may feel crepitus

Digitized by Google

over the fractured rib. You must not examine such a patient standing up, as is usually done. Now the reason why diagnosis of fractured ribs is often difficult is this: the fragments of rib are often more or less held in position by untorn periosteum, and therefore they do not grate upon one another. Again, ribs are often thickly covered by muscle; so that without the aid of the X rays it is not always possible to be certain that fractured rib exists. But the great pain on breathing or coughing, and on local pressure, is a strong inferential symptom. The ordinary treatment for fractured ribs is to strap and bandage the chest. Broad pieces of strapping are applied from below upwards, and reaching from the middle line behind to the middle line in front. These are placed on during expiration. Then a broad bandage is applied from below upwards. This, if properly done, gives great relief. I may add, it is a simple matter, and seldom consequently studied by students. Fractured ribs join by "provisional callus," the reparative material being thrown out round the bone as a kind of ferrule. This external callus becomes absorbed as union of the fractured ends progress. So that before a broken rib is firmly joined by bone a very long period must elapse, and this period is very variously stated by different authors.

The leading symptoms of injury of the lung in a case of fracture of the ribs are as follows:— First there is emphysema of the chest wall. If there is a sensation of crackling produced by air in the chest wall, this is a sure sign that the lung is wounded by the fractured rib. The next most important sign is arterial hæmoptysis, the spitting up of bright blood mixed with froth and mucus. The special complication which is likely to follow upon injury of the lung is traumatic pneumonia. The treatment of fractured ribs associated with traumatic pneumonia is the only subject I will now deal with. Injury to the abdominal contents is a larger subject, and time does not allow us to discuss it. Pneumonia being a common and serious complication of fractured ribs, it is very important you should know how to treat it. As a rule, the patient with fractured ribs associated with pneumonia cannot bear to have his chest percussed and over-examined. It is very bad practice to sit such a patient up two or three times a day and percuss over his broken ribs to see how his pneumonia is progressing! The usual signs of pneumonia in such a patient are high fever, a dry burning skin, with a quick pulse, dyspnœa, and especially a dusky colour of the face, which is often more marked on the side on which the pneumonia is present. He tries to suppress his painful cough, and the well-known "rusty" sputa may not appear. If you listen carefully to the chest without hurting the patient, you will hear such signs as fine crepitation or tubular breathing, and with this you may be satisfied without further examination of the chest. The treatment of these cases of pneumonia is usually a little different to what is done in medical cases. Traumatic pneumonia from fractured ribs often occurs in very strong plethoric individuals, such as "hunting men" and farmers. This class of man is usually benefited by two remedies: one is antimony, and the other is venesection. When there is evidence of engorgement of the right side of the heart, as evidenced by duskiness of the face, and venous engorgement, bleeding is very beneficial. In painful traumatic pleurisy with fever, if you wish to earn the eternal gratitude of the patient and his friends, you will relieve his congestion by blood-letting. The flow of blood unloads the right side of the heart, and allows respiration and circulation to proceed. Of course there are medical measures to be used. Expectorants are often exceedingly useful; any of the expectorants in the Pharmacopœia may be employed, and among these may be mentioned ipecacuanha, iodide of potassium, squills, and antimony. Morphia must be administered with great caution to combat sleeplessness. As to what cases you are to give one of these remedies to, and to what class of cases you are to give others to, that is a matter of general medical knowledge. For instance, in London, in the case of a feeble broken-down cabman with fractured ribs and a wound of the lung leading to pneumonia, you give carbonate of ammonia as an expectorant and stimulant, and administer alcohol freely. But in a man who is strong and plethoric, an inhabitant of the country, you can give antimony and bleed him. I recommend you to remember these remarks about fractured ribs, because the accidents are common. You are sure to meet with them, and therefore you must know how to treat them. I ask you to always observe the very simple remedy which we have applied here,

Digitized by Google

and which is too often ignored. Keep the patient in the sitting posture. That simple precaution may save many a life after broken ribs, by warding off congestion and consequent pneumonia.

I next wish to speak about another exceedingly common accident of general practice—fractured humerus. In this present case the fracture of the humerus is about the centre of the bone. There are so many ways of treating this common accident that I cannot mention them all now. I will merely relate the method which I adopt myself after a good many years of practice in these cases. If the humerus is fractured at or about the middle of the shaft, a useful method is to place the limb upon an inside angular splint, taking in the elbow, and place some short splints externally and in front. If you are in reach of a skilled instrument-maker, you can get him to make a leather splint, which I think is the best for these cases, with a cap taking in the shoulder and elbow. The two portions of the splint can be laced together, and they can be undone and the arm daily massaged. Such a leather splint well made, combined with massage, is the best ordinary method of treating fractured humerus when the fracture is in the middle of the shaft. It has long been my own practice in fracture of the humerus near the surgical neck, as soon as the swelling and œdema subsides, which it usually does in about a fortnight, to get an assistant to hold the fracture in position by extension, and set the shoulder in a plaster-of-Paris bandage reaching from below the elbow up the entire arm. This is taken off in about five weeks' time, and massage is well applied to the shoulder and elbow. This is one of the few fractures in which I would employ the plaster of Paris. And I may say that the nearer the fracture occurs to the surgical neck of the humerus, the more is such an application required. No splints are of avail in such cases as these. There is a very awkward complication, which probably is in the minds of some of you, about the fracture of the middle or upper third of the humerus; it is injury of the musculo-spiral nerve, or the implication of that nerve in the subsequent callus or inflammatory effusion about the fracture. Surgeons who are unlucky enough to have a case in which "wrist drop" occurs will receive a good deal of blame which they do not deserve! No doubt many of you have also in your minds the question of wiring together fractures of the humerus. Wiring a fracture of the humerus is an easy operation compared to wiring a fracture of the femur, for in the case of the humerus the bone is fairly superficial, and there is no reason why wiring should not be adopted in cases of very troublesome oblique fractures, especially high up. If you take a skiagram of the fracture, which you should do, and you find the bones seriously and persistently misplaced, it would be justifiable to cut down and wire the fragments together. My own opinions about these operations are well known to you. I think such treatment is only exceptionally necessary. Now there was a complication in this case, one which often occurs in cases of fracture in the so-called "working" classes about Christmas time; I refer to alcoholism. We were able to keep this man quiet by means of bromides, but in cases of alcoholism and fracture, where the patient is the subject of delirium tremens, where he throws his arms about, delayed union is apt to cause anxiety in the case.

Next we will consider the case of a man about forty-five with a subpectoral abscess. Most of you would probably be inclined to pass him by on the other side as not likely to be the kind of case asked about in examinations, but the affection is very common, and therefore very important in ordinary practice. So we will spend some time over it. This man got a poisoned finger, a septic wound, with consequent inflammation and suppuration in the glands above the internal condyle of the humerus, and in the axilla. Some of the worst of these cases are found in members of your own profession through conducting post-mortem examinations or attending a patient with poisoned wound. So this affection is common in a man who follows agricultural operations; he may get a prick on the finger from a thorn or thistle. If you practise at the sea-side you will hear of or see fishermen who have had a septic wound inflicted by a dirty fish-hook covered with putrid material. Such injuries occasion violent inflammation. Remember the pus forms underneath the fascia; consequently there is no fluctuation, only a "brawny" tense swelling. If, as is often erroneously done, you wait until fluctuation is evident, you wait until the whole pectoral region is dissected out by the abscess, and the function of the arm for the future considerably impaired. Therefore the right practice in these cases is as follows:—When you feel a

Digitized by Google

brawny swelling in the axilla and the patient suffers great pain and fever, you should explore for pus. Next mark this special point. The safe place to make the incision is on the inner wall of the axilla, on the thoracic wall, midway between the anterior and the posterior flaps. Cut through the fascia there, and then break into the abscess with your finger, which is far better than any instrument where large vessels are near. Put in a large tube and drain the cavity. If you open these abscesses freely and early in this way they will soon heal, and then movement and massage can be carried out with good results. This patient, you see, is a poor, miserable-looking, ill-nourished man, and I fear that before we get him well we shall have to lay the sinus open. It was long neglected, and the pus has "burrowed" seriously. I need hardly say that the opening should be done under a general anæsthetic. Another point about these abscesses is as follows:—Septic organisms or germs may lie latent in the axillary glands for a long time, and finally cause suppuration; I have known an instance where a surgeon in the west end of London pricked his finger at a post-mortem and got enlarged painful glands in the axilla. But these glands did not suppurate till eleven months afterwards! When the pus was examined bacteriologically it was found to contain *Staphylococcus aureus*, and I have no doubt the micro-organisms had been in the glands all that time. The treatment, then, of subpectoral abscess is summed up in operating early, opening the inner wall of the axilla, and draining freely, doing this deliberately under a general anæsthetic. Avoid making small "pricks" under gas, or attempting to operate without anæsthetics!

This next case well illustrates the extraordinary difficulty one sometimes finds in the diagnosis and treatment of appendicitis. The patient is a man æt. 47. A month before admission he felt a sudden acute pain over the abdomen. It was vaguely distributed; he could not localise it in the least. Afterwards he localised the pain to the right side of the abdomen. To complicate matters, about ten years ago he had a lumbar abscess which opened on the buttock, the scar of which was visible, but the history and causation of which was quite obscure. When he came in he looked ill, but his temperature was normal. It afterwards rose. The only thing one could detect was a slight hard swelling deep down in the right iliac fossa. The muscles were not rigid, and they moved on respiration. This swelling felt very deep, and its true nature was absolutely dubious; one could not be at all certain of its nature. Nothing could be felt *per rectum*. But seeing the old scar over the buttock and hearing the story of an abscess ten years ago, one naturally began to suspect that it was a recurrence of the old mischief in connection with diseased bone in the pelvis. After due consideration, I resolved to make an exploratory opening into the swelling and see its nature. I cut down in the iliac fossa and turned the intestines and cæcum back, and found a very firm swelling just behind the cæcum, reaching up towards the kidney. On palpating this with my finger I broke through a membrane into a cavity, which presumably contained pus. I kept my right finger in the opening to prevent the pus running back into the general peritoneal cavity, and with the left hand made a counter opening in the loin, coming upon a large foul fæcal abscess which tracked up to the kidney. I felt a long disintegrated appendix, which with the aid of both fingers I was able to shell out. It was one of those appendicitis cases in which the abscess burrows up behind the cæcum and passes towards the kidney. This very obscure case was an instance of appendical abscess caused by perforation of a retro-cæcal appendix which passed up towards the kidney. With regard to the after-treatment of cases of this nature, very careful drainage has to take place through the lumbar wound by means of a large tube. The bowels have to be relieved shortly after the operation, because they frequently get distended. This man was in great distress for a couple of days after the operation, but by the use of the old-fashioned rue enema which is often used in this hospital, coupled with the administration of calomel, we managed to get rid of the flatus and relieve him.

The next case which I specially want to draw your attention to is a large fluctuating swelling in the right groin, which has a distinct impulse on coughing, and which is reducible under Poupart's ligament. But observe that it does not go back suddenly like a hernia, but gradually. There is no gurgling of gas in it on palpation. The next point is as follows:—When this fluid is pressed back beneath Poupart's ligament, it fills up a swelling deep in the iliac fossa. You can press

Digitized by Google

the fluid out into the thigh again, and thus you are able to get fluctuation from hand to hand beneath Poupart's ligament, which you see I am now doing. This is the great test in differentiating psoas abscess from hernia, that the pus fills the iliac fossa and you get fluctuation from hand to hand. Again, abscess is dull and hernia is resonant to percussion. Moreover, frequently, as here, the abscess is not in the exact situation of a hernia. The common cause of chronic abscess passing down into the thigh is tuberculous caries of the spine. Caries of the spine may be very ill marked in these cases. It is by no means necessary always to get curvature and distinct signs of spinal disease. The caries may be on the side or in front of the lower vertebræ, but the symptoms are so vague that the disease is only verified by autopsy. In no cases are so many diagnostic errors made as in spinal caries, especially in adults. The methods of treatment in a case like this are very various. The common practice is to make an opening into the abscess and let out the pus, then wash it out, put in a large tube, and drain antiseptically. You have heard me often speak on the practical difficulties which attend keeping these large abscesses in the groin aseptic. When there is much discharge these difficulties may be almost insurmountable, especially in private houses. The practice I adopt in "cold" abscess of this nature is as follows:—I either treat the abscess by repeated aspiration, or by opening and washing it out, putting in a little iodoform emulsion and sewing the incision up again. I lightly "curette" the walls of the abscess with a blunt spoon, and flush out the *débris* very carefully. A prolonged residence in sea air is very beneficial. If once you open and drain these large abscesses, and the cavity becomes infected, a hectic temperature comes on and persists, and the patient is very likely to ultimately die.

The next case is an example of the serious complications which are sometimes associated with acute gonorrhœa. This unlucky man infected his eye with gonorrhœal pus, and you will see he has had extensive ulceration of the cornea with opacities. This is not all. During the acute stage of the attack he got a bad septic inflammation of the knee. You know the affection which is termed gonorrhœal rheumatism,—in other words, infection of the synovial membrane by the gonococcus. Commonly this complication occurs about the fifth or sixth week of the disease. But in some bad cases it occurs early, and then you may practically look upon the joint as affected with pyæmic inflammation. It is full of sero-pus and masses of yellow purulent lymph. The treatment of these acutely inflamed gonorrhœal joints, which I am sorry to say I have myself adopted, was to rest them in splints, with the application of all the remedies which were known to subdue inflammation, such as ice, leeches, and the like. The joints then absolutely stiffened, tough adhesions formed, the cartilages became more or less destroyed, and the best result you could expect to get was firm ankylosis. But for the last two or three years I have adopted a method which has given remarkable results. I have opened the joint by an incision about one inch long, and washed it freely out, using mercuric biniodide solution 1 in 1000. The pain has at once been relieved, and the fever has subsided. The wound is united, and when healed massage is adopted. The cases which I have hitherto so treated have been five in number. They have recovered with useful mobile joints. If you look at the temperature chart in this man's case, you will see there was a distinct drop after the operation. There is great thickening of the peri-articular structures in this patient, and I advise that the rubber should go energetically to work on the case, and not be afraid of a little pain being caused. Together with the treatment of the knee, the urethra should be syringed out with biniodide of mercury lotion. The best remedies to give internally are iron and quinine. I think the term "rheumatism" should be abolished in these cases. We are all too apt in practice to look upon swollen joints as evidences of "rheumatism," and we should remember that such conditions may be due to a variety of causes.

This woman is a very important case, a cyst of the breast closely simulating carcinoma. Near the nipple was a hard lump about the size of a filbert-nut, and there was deep retraction of the nipple, which you know is rightly looked upon as a very important sign of carcinoma. The contracting cancer pulls upon the milk-ducts and fibrous tissue which passes to the nipple. Retraction of the nipple is looked upon as so strong a symptom of carcinoma that many surgeons, if they feel a hard

Digitized by Google

lump and retracted nipple, will pronounce the tumour as carcinomatous straight off. I have drawn attention in literature to several conditions which may contract the nipple other than cancer. A deeply seated chronic abscess in the breast will contract and pucker the nipple, and so will a shrivelled cyst. Varieties of chronic mastitis will also cause the same symptom. In this case the little lump was so hard and the nipple so retracted that I thought, I must confess, the affection was cancerous. We discussed the question also of the swelling being a cyst filled with solid growth. However, in this opinion we were wrong. When I cut into the swelling there came out turbid fluid. It was simply a cyst about the size of a filbert, with unusually thick walls. It was dissected out and the breast saved. This is another illustration of what is so important in the case of small hard tumours of the breast. It is unwise to pronounce a definite opinion regarding the nature of such tumours until they have been explored. This is an important practical rule. Hereafter, when you are in practice, you will find a lady will come to you in great alarm, having discovered a small hard lump deep in the breast, which causes her mind to at once dwell upon cancer. She will ask you definitely, "Is it cancer or not?" In the case of these small deep-seated tumours in the breast it is only the confident and ignorant man who will give a decided opinion without making an exploratory incision. It is far better to say under such circumstances, "You should have an operation immediately done so that the true nature of the swelling may be ascertained." A large number of these deep hard lumps in the breast turn out to be cysts, though they may closely simulate cancer. Some of you may recollect a woman in an adjacent bed who was a nurse, and who had a tumour of the breast about the size of an orange, firm, deeply seated, and of rapid formation. The opinion expressed after consultation was that the tumour was a sarcomatous or quickly growing cancerous growth. I rather inclined to the same view, and made an exploratory incision. We found a large cyst as big as an orange filled with turbid fluid, and without any growth in it whatever. I was able to dissect it out, and there was no further trouble. Remember that if you make a mistake in these cases and tell your patient she has got cancer when she has only a cyst, or if you do the reverse and tell your patient that she has not got cancer but only a cyst when the swelling is really cancerous, you will be severely blamed. Remember that these cases are eminently suitable for consultation and exploration, and never be too positive. Such instances will be brought before you in practice as surely as you now stand round this bed. You will do well to recollect that retraction of the nipple does not always necessarily mean that there is cancer present.

Here is a woman with an ulcer of the skin of the face. It is an exceedingly important case. She is æt. 40, and the first point I want to draw your attention to is that there are really three small ulcers and not one. There is one under the eye, one close to the right of the nose, and another lower down. This in itself is almost a diagnostic point against epithelioma. Multiple epitheliomatous ulcers are exceedingly exceptional. I once published a case in connection with undue exposure to the rays of the sun. Multiple epitheliomata may also occur with a rare skin disease which you may never see in a life-time. When you feel the edges of these ulcers you find they are distinctly soft. That also is against the diagnosis of cancer. Another fact against epithelioma is the time during which the woman has had these ulcers. The total duration of them is six months. Epithelioma would hardly grow to this extent in six months. In the case of epithelioma there would first of all have been a pimple or wart on the face, or some premonitory cause of the disease, and you may take it as a valuable rule in practice that ulcers of the skin of the face which progress rapidly are generally specific in nature,—that is to say, tertiary or late manifestations of syphilitic disease. This I imagine is the true explanation here. Additional evidence of this view is the circular or serpiginous outline of the ulcers. You may ask me, Are there any other possibilities? Well, it may be a tuberculous infection of the skin of the face; therefore it will be worth while to submit the discharge to the bacteriologist for cultivation to see whether it contains any tubercle bacilli. But such an examination must not be too strictly relied upon. The well-known rodent ulcer of the skin of the face you may put out of court. The growth has been far too rapid for that. Rodent ulcer to reach this stage would take something like six years rather than six months. The varieties of ulceration of the face which are common I have enumerated to you. The more rapidly the ulcer

Digitized by Google

extends, the more serpiginous its outlines, the more likely it is to be due to syphilis. Epitheliomatous ulcers begin as a pimple or wart, having hard indurated edges. The glands may or may not be enlarged. We shall give this woman iodide of potassium and perchloride of mercury, and increase the doses gradually, at the same time using the following ointment, which is very valuable for syphilitic ulcers,—namely, the red oxide of mercury ointment one drachm to one ounce of lanoline. The red oxide of mercury ointment is most valuable for these cases if it is not applied too strong. Dressings with this application should be kept constantly applied. If we discover that with this treatment the ulceration does not heal, and if we find tubercle bacilli in the discharge, the proper treatment will be to freely destroy the ulcer with a curette and with caustics. The best caustic is, I think, "potassa fusa." Remember that the evidences of old syphilitic disease in women, and especially married women, are exceedingly vague. In them you can very seldom get any distinct history, for the simple reason that the patients do not understand anything about the infection. And it may be that the signs of infection have been so slight that they never noticed them. So in any class of society, whenever you get these curious ulcers on the face remember that syphilis may be the true cause. These cases are easily confounded with cancer, and reputations easily wrecked regarding them.

We have received from Messrs. Burroughs, Wellcome & Co. a sample tube of 'Soloid' 'Hemisine,' presenting the hæmostatic principle of the supra-renal gland in a stable, soluble, and convenient form. The various advantages of this "soloid" in the treatment of certain conjunctival disorders, in the control of bleeding, in allaying the symptoms known as hay fever, and in other ways should be well known to practitioners. It it interesting to note how therapeutical experience is affording proof of the truth of the theory, founded on pathological grounds, that the medulla of the supra-renals produces an internal secretion which it imparts to the blood. The prostration in Addison's disease is reasonably ascribed to the want of this secretion, though the sickness and pigmentation have not as yet been accounted for.

TWO CLINICAL LECTURES

ON

ANEURYSM OF THE ABDOMINAL AORTA.

Delivered at Guy's Hospital, July 8th and 15th, 1903.

By J. H. BRYANT, M.D., F.R.C.P.,
Assistant Physician, Guy's Hospital.

Lecture I.

Gentlemen,—I have already, in another lecture, alluded to the rarity of this disease, and will again refer to the figures on which this statement is based. I analysed the reports of the post-mortem examinations which have been performed at Guy's Hospital between the years 1854 and 1900 inclusive. During these forty-seven years, there are records of 18,678 necropsies, and of these, 325 were cases of aneurysm of the aorta, of which 54 were of the abdominal portion.

It has been stated that the abdominal aorta is only affected by aneurysm in 5 per cent. of the cases, but the above figures indicate a higher frequency, viz. 16 per cent.

Ætiology.—Males are much more liable to this disease than females, for of the 54 cases 49 were men, *i. e.* over 90 per cent.

The ages were as follows:

Under 20 years	2
Between 21 and 30	11
Between 31 and 40...	23
Between 41 and 50...	8
Above 50	10
	54

So that 34, or 63 per cent., occurred between the ages of twenty-one and forty; and 44, or 77 per cent., between twenty-one and fifty.

This analysis does not confirm the statement that it is a disease of middle age, when of these 54 cases 36, or 63 per cent., occurred before the age of forty. These figures, however, correspond to the analysis of the cases collected by Crisp, of whose 59 cases 33 were under forty years of age.

The two main factors in its causation are syphilis and violent exertion. In many cases these two causes are associated, for syphilis predisposes to the condition by causing a local weakness of the artery, and the increase of blood-pressure resulting

Digitized by Google

from a sudden strain or violent muscular exertion causes a bulging of the wall of the artery at the weak spot. The fact of it being so much more prevalent in men than in women points to the probability of some marked difference of environment, which is at once evident if their respective occupations are contrasted; and further, syphilis, the all-important predisposing cause, is much more common in men than in women.

A few days ago I was discussing the question of aneurysm with my colleague, Dr. Pitt, and he told me that in the Newcastle-on-Tyne district, where aneurysm of the aorta is far from being uncommon, Prof. Drummond, who has been carefully investigating the ætiology of this disease, has come to the conclusion that in practically all the cases the patients have at some time suffered from syphilis,—in fact, the presence of an aneurysm of the aorta may be looked upon as almost conclusive evidence of syphilis.

It is far commoner in the labouring classes than the well-to-do, for men of the former station in life follow the occupations of porters, labourers, engineers, sailors, lightermen, etc., and have to earn their living by engaging in hard manual labour, which often entails sudden and violent muscular efforts. It may also follow injuries to the back or abdomen. The following are some of the exciting causes to which it has been attributed:

(1) A man æt. 34, who seven years before had contracted syphilis, after lifting a heavy weight experienced severe pain in his abdomen, which was followed by sickness and a sensation of throbbing.

(2) An artilleryman æt. 30, who had suffered from syphilis, experienced a severe pain in his back whilst helping to lift a heavy gun with a lever, and after this the pain in his back became severe and constant.

(3) A man æt. 33, who had suffered from venereal disease, slipped on the pavement and strained himself. This was followed by a severe and constant pain in his abdomen.

(4) An engineer was carrying a heavy iron axle, when he suddenly turned round and ricked his back, and at the same time experienced a sharp pain in his loins.

(5) A man æt. 46, employed at the military stores, about eight years before coming under observation, after running a race, had a severe fall, and stated that he was "never afterwards strong in his loins, and suffered from pain in his back."

(6) A porter æt. 34, accustomed to carry heavy weights on his back, strained himself about three years before admission to the hospital, and had since suffered from pain and pulsation in his epigastrium.

(7) A wood-carver æt. 38, who died from the rupture of an abdominal aneurysm into the retro-peritoneal tissues, had been accustomed when a young man to violent boating exercise.

(8) A lighterman æt. 28, who had suffered from syphilis ten years before admission, had frequently strained himself at his work.

(9) A draper's assistant æt. 28, eight months before admission fell down a staircase and injured his back. Two months afterwards he was seized with pain and stiffness in his left side.

(10) A ship's painter æt. 36, who fifteen years before admission had suffered from syphilis, and eleven years afterwards fell 46 feet, striking his back across a log, died from rupture of an abdominal aneurysm.

(11) An army labourer, who had served in the army, and whilst in India contracted syphilis, for which he was treated for forty days only, had been engaged in lifting heavy shot, when he first noticed a swelling of his right leg. He subsequently died from the effects of an aneurysm at the bifurcation of the aorta.

In many of the cases there is no direct evidence of an actual exciting cause, but there is usually a history or evidence of syphilis, *e. g.*—

(12) An engine-driver æt. 33 was admitted for pain in the lower part of the back. Seven years before he had contracted syphilis, and the first indication of the presence of an aneurysm of his abdominal aorta was a gnawing pain in the back, which commenced six years afterwards.

In twenty out of the fifty male cases there was a history of venereal disease, and in fifteen there appeared to be no doubt that the disease had been syphilis.

Many of the earlier reports were meagre, so that no reliable statistics as to the frequency of syphilis can be adduced from this series. The most striking feature elicited from the analysis was, that eight of the fifty males had served in the army, and all had suffered from syphilis.

I will take this opportunity of drawing your

Digitized by Google

attention to the scandalous manner in which syphilis has been treated in the army. For several years I have always made a point of inquiring from patients who have contracted syphilis, whilst in this service, of the manner in which it had been dealt with, and I have found that almost without exception, after a few weeks in hospital, when all external manifestations of the disease have disappeared under the influence of mercury, the soldier is sent back to duty, and goes untreated until some secondary or tertiary indication appears, when a further perfunctory and inadequate course of treatment follows.

One of the most noticeable facts brought out by an analysis of these fifty male cases was, that eight had served in the army, and all had suffered from syphilis whilst on service. I repeat that it is a grave and scandalous state of affairs that syphilis, a disease which is universally recognised as necessitating for eighteen or twenty-four months careful, and more or less continuous treatment, should be dealt with in this disgraceful and slipshod manner in the army. It is degrading to the profession that the medical officers should so pander to red tape in such a vital question, and the sooner the matter is investigated and dealt with in a proper manner, the better it will be for this and future generations.

The fact that the eight men who had served in the army had all had syphilis, and had subsequently developed this very rare and fatal disease, is additional proof of the inadequate treatment they received. The history of the patient described above, who was recognised as suffering from syphilis, and was treated for forty days only, is typical of the method which has been in vogue in the army for the treatment of this terrible disorder.

Next to syphilis acute rheumatism and chronic alcoholism appear to be the commonest associated diseases, but whether the changes produced by these conditions have any influence in causing the disease is not evident.

VARIETIES.—There are five varieties of aneurysm of the abdominal aorta, viz.—saccular, diffuse, dissecting, fusiform, and arterio-venous. Of these the saccular and diffuse forms are by far the commonest, and the arterio-venous the rarest, there being only two of this last-mentioned kind out of the fifty-four. The diffuse form is usually produced by the rupture of a saccular aneurysm into the adjacent tissues. The leakage of blood may be gradual, but surely progressive, until a large tumour is produced, which may displace and press on adjacent viscera and structures.

The saccular and diffuse forms may arise from any part of the vessel, but most frequently spring from the neighbourhood of the orifice of the cœliac axis. An analysis of the fifty-four cases shows—

36, *i. e.* 67 per cent., in the neighbourhood of the cœliac axis:

11 at the orifice.
13 above the orifice.
12 below the orifice.

5 at the orifice of the superior mesenteric artery.

3 at the orifice of one of the renal arteries.

All the above were saccular or diffuse. Fusiform aneurysms involve the lower part of the vessel; dissecting the whole of the abdominal aorta and often a part or the whole of the thoracic aorta also; the arterio-venous, the lowest part, at or just above the bifurcation. The sac may arise from the anterior, lateral, or posterior aspects of the vessel, and may vary in size from a slight local bulging to a tumour which measures several inches in diameter.

Erosion of the bodies of the vertebræ is one of the chief effects of the gradual increase in the size of the sac—a condition which is more likely to occur when the aneurysm arises from the lateral or posterior wall of the vessel. The amount of destruction may vary from a slight and superficial erosion to an almost complete destruction of a body or bodies of the lowest dorsal and upper lumbar vertebræ, leading to exposure and pressure on the spinal cord and its nerve-roots. A curious but constant feature of this erosion of bone is the invariability with which the intervertebral cartilaginous discs escape destruction—a very striking contrast to the effect of spinal caries from other causes, in which the discs are usually the first to suffer, and to be more extensively destroyed than the adjacent bone. This erosion occurs in about 40 or 50 per cent. of the cases. It depends entirely on the direction in which the sac of the aneurysm enlarges.

Aneurysm of the abdominal aorta eroding the vertebræ and rupturing into the peritoneal cavity.—A man æt. 89, who had been a seaman, was admitted on October 23rd, 1867, under the care of Dr. Rees, for pain in the back over the lower dorsal vertebræ. Four years before admission he began to suffer from pain in the epigastrium, and

Digitized by Google

a pulsating abdominal tumour was discovered. On admission he complained of pain over the ninth, tenth, and eleventh dorsal vertebræ, the spines of which were protruded, and there was a pulsating abdominal tumour in the region of the cœliac axis, over which a systolic bruit was heard, both in front and over the corresponding part of the back. He died suddenly on November 28th, having suffered from severe pain and insomnia.

Post-mortem examination.—Sixty ounces of blood-clot were found in the peritoneal cavity. There was an aneurysm of the aorta, which implicated the whole of the calibre of that vessel for about 4 inches, which had ruptured just above and to the right of the pancreas. The sac had thrust away the diaphragm, pancreas, and liver from the spine, and posteriorly had eroded the bodies of the eleventh and twelfth dorsal vertebræ, a laminated clot filling the space between the vertebræ and lying in contact with the dura mater, but not causing any pressure on the cord. The pressure on the vertebræ had been from the front and also from the sides. On each side of the spine the aneurysm extended for 2 or 3 inches, and pressed on the orifice of the nerves.

If death occurs as a direct result of the aneurysm, rupture is almost always found as the cause.

Of the fifty-four cases,

Rupture into the	retro-peritoneal tissues occurred in	19
,, ,,	peritoneal cavity occurred in	12
,, ,,	pleural cavity occurred in	8
,, ,,	mediastinum occurred in	2
,, ,,	pericardial sac occurred in	1
,, ,,	mesentery occurred in	1

Rupture has been known to occur into the stomach, duodenum, lung, pelvis of the kidney, bladder, gall-bladder, or bile-duct. When the sac ruptures into either of the serous cavities an enormous quantity of blood and blood-clot is found, and in the case of the pleural cavity the corresponding lung is compressed and airless, and the heart is displaced to the opposite side of the thorax.

If the rupture occurs into the retro-peritoneal tissues, the effused blood pushes forwards the peritoneum, and the structures which lie in front of it become displaced; the kidney may be pushed forwards and downwards; the ureter may be kinked or obstructed with hydronephrosis as a consequence; the duodenum may be compressed, twisted, or kinked, with dilatation of the stomach as a result; the pancreas and other viscera may also have their normal relations altered. The blood may plough up the substance of the psoas and quadratus lumborum muscles, and burrow downwards into the thigh and backwards into the loin. In one case the genito-crural nerve was dissected out by the blood-clot. If the blood extravasates behind the diaphragm into the posterior mediastinum, it may compress and obstruct the œsophagus, and may even extend upwards and cause a swelling in the neck. The inferior vena cava may be displaced or pressed on by the sac of the aneurysm, but it is by no means a common result. A communication between the aorta and inferior vena cava may form, but it only occurred in two of the cases, in both of which the aneurysm involved the aorta at its bifurcation. Œdema of the legs and lower part of the body may be produced if the inferior vena cava becomes obstructed, and is particularly well marked and is associated with the presence of enormous distension of the superficial veins of the leg, scrotum, and lower part of the abdomen in cases of arterio-venous aneurysm.

Aneurysm of the abdominal aorta opening into the inferior vena cava.—A paperhanger æt. 28, was admitted on January 2nd, 1880, under the care of Dr. Wilks, for abdominal pain and vomiting. Seven years before he had contracted syphilis. Eight weeks before admission he was treated for bronchitis, but did not improve and began to suffer from pain in his abdomen, bowels and loins, and also from severe vomiting. Three weeks ago his legs and abdomen began to swell.

On admission he found it easier to sit up in bed than to lie down. His legs were swollen and œdematous. A loud, systolic bruit could be heard along the course of the aorta, all over the abdomen, and down the outer side and the back of the thighs. The diagnosis was constriction of the descending aorta, either from a tumour in the posterior mediastinum from disease of its walls, or from a deep aneurysm pressing upon it. On January 26th he was examined by the late Dr. Hilton Fagge, who stated that the bruit was loudest in the left lumbar region. On March 3rd Dr. Wilks regarded the case as one of aneurysm of the abdominal aorta compressing the inferior vena cava. On March 8th the legs and scrotum were

Digitized by Google

so œdematous that acupuncture of the legs was performed, and about seven pints of fluid drained away. The superficial veins over the abdomen were very much distended, and formed a complete network. On March 11th inflammation was noticed around the punctures, and on the 13th the right leg became gangrenous. He gradually became worse, and died on the 20th.

Post-mortem examination:—The right leg was very gangrenous in appearance. The veins all over the trunk and thighs were enormously distended, and in places there were distinct varicosities. There was a little atheroma of the thoracic aorta. The abdominal aorta was healthy until just above the point of bifurcation, where it was very atheromatous, and was dilated on the right side into an aneurysmal sac, which also involved the upper part of the right iliac artery. The aneurysm was quite empty. It pouched towards the right and front, but chiefly to the right, and had become adherent to the lower end of the inferior vena cava, the coats of which were yellow and diseased-looking over the area corresponding to the sac of the aneurysm. In the upper and right part of the aneurysm there was a vertical oval aperture with thick smooth edges, which opened directly into the inferior vena cava. The coats of the vein and artery at this spot and around it were thickened and perfectly continuous and adherent, and through the centre was the aperture.

The renal veins may be compressed and thrombosed, and the kidneys become congested and ecchymosed. In one case blood was effused into the right spermatic cord, and the corresponding half of the scrotum was much ecchymosed. If the spermatic artery is obstructed, atrophy of the testicle follows. When the sac arises from the lower part of the aorta one of the iliac veins may be compressed, and œdema of the corresponding leg is the result. An aneurysm arising from the right side of the upper part of the aorta in the region of the cœliac axis may press on the portal vein and bile-ducts and give rise to ascites and jaundice.

In more than 50 per cent. of the cases it is customary to find ante-mortem thrombus in the aneurysm, which may vary from a thin layer to a laminated mass completely filling the sac, a result aimed at by the various methods of treatment. As one or more of the branches of the abdominal aorta are generally found to arise from the sac of the aneurysm, one of the effects of the formation of an ante-mortem thrombus is in some cases to block these vessels. One of the renal arteries is most frequently affected in this way, and if it has been obstructed for some time the kidney is found to be atrophied. In one case in which the right renal artery was thus obstructed, all that could be seen of the right kidney were a few shrivelled fibrous remains which measured less than three quarters of an inch in length. The blocking of arteries in this manner is usually a gradual process which allows time for the formation of a collateral circulation, or the results would be extremely serious, and similar in effect to those produced by embolism, especially if the superior or inferior mesenteric arteries were the vessels implicated.

In one case the aneurysm commenced immediately below the cœliac axis, and the orifice of the superior mesenteric artery was carried forwards and pressed on by the sac, and it also came from a part of the sac which was filled with fibrin, so that it was completely obstructed, and no blood could have gone by the direct route to the intestines supplied by this vessel. On the other hand, there was a remarkable large tortuous vessel running down to the superior mesenteric in the position of the colica transversa, and it had evidently received its blood by the colica sinistra of the inferior mesenteric, which artery was abnormally large.

The thrombus in an aneurysmal sac may gradually increase in size until it finally blocks the lower end of the aorta, or pieces of the thrombus break off, and embolic blocking of the renal, superior mesenteric, inferior mesenteric, iliac arteries, or even the aorta itself, follows as a result. Necrosis of the kidney may arise from embolism of the renal artery; infarction and sloughing of the intestine from embolism of the mesenteric arteries; gangrene of the legs from embolism of the iliac arteries or lower end of the aorta. In most cases atheromatous changes are found throughout the whole of the aorta, but on the other hand, with the exception of the portion involved by the aneurysm, the vessel may be perfectly healthy—a condition which is found more frequently in young than in old people.

Peritonitis is a very rare complication, and only occurred in those cases which had been treated by compression. If an aneurysm arises from an apparently healthy-looking aorta, it is usually the

Digitized by Google

result of an injury which has led to damage or rupture of the intima.

Symptoms.—Pain is the most constant and usually the first indication of the presence of an aneurysm of the abdominal aorta. It does not depend on the position of the sac, and is extremely variable in its site, character, duration, and severity. It may be constant and uniform, or intense and paroxysmal. It is usually referred to the back or abdomen, especially to the epigastrium, or may be referred to the loin, groin, hip, sacro-iliac region, or chest. It may extend round the body, or shoot down into the legs or testicles. It was described by different patients as severe, continuous, dull, heavy, aching, throbbing, boring, gnawing, tearing, or neuralgic. Pain which has been dull and aching may suddenly become intense and paroxysmal in character. Pain is generally associated with erosion of the bodies of the vertebræ, but it does not necessarily follow that it is always the cause, for in some cases, in spite of the most extensive destruction of bone, the pain is slight. On the other hand, the pain may be very severe and paroxysmal in character without the existence of the slightest erosion of the vertebræ, and it is under these circumstances most probably due to the stretching of nerve filament over the sac. Dr. Habershon has recorded an interesting case which illustrates this point. The patient suffered the most intense pain, and at the necropsy a small aneurysmal sac was found with no erosion of the vertebræ, but with a network of branches of the sympathetic nerve stretched over it.

If the erosion is extensive, nerve-roots may be compressed and the pain is intense, and corresponds to the distribution of these nerves. Diffuse aneurysms may implicate various nerves—such as the last dorsal, when the pain is experienced over the crest of the ilium ; the genito-crural nerve, with pain shooting into the testicle ; the sciatic nerve or anterior crural, with the pain down the back and front of the leg. Pain resulting from destruction of the bodies of the vertebræ is aggravated by pressure, and by extending or compressing the spine.

If the ureter is pushed forwards and stretched, severe attacks of pain which commence in the loin, radiate in the direction of the ureter to the testicle, penis, and upper part of the thigh are produced, and may be mistaken for renal colic due to the presence of calculi. Whatever the character of the pain may happen to be, it is almost invariably increased by movements of the body, by walking, and by certain attitudes which vary according to the position of the aneurysm. Some patients cannot lie on the left side, others cannot keep still, and have to be constantly shifting their position, whereas others cannot remain flat on their back, and some cannot sit up. As a general rule, the attitude of greatest comfort is sitting up in bed supported by pillows with the body bent slightly forwards.

Insomnia is a very common and troublesome symptom, and is one of the results of the severe pain ; it occurs in more than a third of the cases.

Retching, vomiting, and flatulence from pressure on the gastric branches of the solar plexus, or from actual pressure on the stomach and duodenum, may be present, and the symptoms of dilated stomach may become manifest if the pylorus or duodenum is compressed. Dysphagia may be produced if the aneurysm or effused blood presses on the cardiac end of the stomach or lower end of the œsophagus.

If the aneurysm ruptures into the stomach or duodenum, profuse and fatal hæmatemesis and melæna follow. Hæmatemesis may also occur as a result of embolism of the gastric arteries.

Constipation is another common symptom ; it was noted as having occurred in sixteen out of the fifty-four cases.

Jaundice and ascites may be caused by pressure on the bile-duct and portal vein.

If the aneurysm pushes up the diaphragm and compresses the lung, dyspnœa is produced, and very sudden and urgent dyspnœa follows when the sac ruptures into either pleural cavity. Cough is another common symptom.

One of the earliest symptoms may be a sense of discomfort and throbbing in the epigastrium, and occasionally the patient himself is conscious that he has a pulsating abdominal tumour, and consults his doctor for this reason.

Loss of power and sensation in the legs and involuntary passage of urine may occur as a result of extensive erosion of the bodies of the vertebræ, with consequent pressure on the spinal cord.

Digitized by Google

If the aorta becomes obstructed with a large embolus, or by the extension of a thrombus from the sac—conditions which produce anæmia of the spinal cord and a sudden cutting off of the blood-supply to the muscles and nerves of the lower part of the body,—somewhat similar symptoms to those caused by pressure on the cord may occur.

Aneurysm of the abdominal aorta; embolism of the aorta; gangrene of the legs; peritonitis; partial paraplegia.—A glass-cutter æt. 40, was admitted under the care of Mr. Cooper Foster on December 4th, 1874, for weakness and gangrene of his legs. He had suffered from syphilis. On October 26th he noticed when he got out of bed a tingling sensation, and a difficuity in moving his legs. Loss of power and impaired sensation followed. He also lost control of his bladder and rectum.

On admission there was diminished sensibility of both legs and feet, and a considerable loss of power, especially in his left leg. The tip of the right toes presented a black and gangrenous appearance. There was neither tenderness of his spine nor pulsation in his femoral arteries. There was no abdominal tumour, but marked pulsation was felt in the right hypochondriac region. A trace of albumen was found in the urine. He became delirious, had incontinence of urine, and gradually sank and died on January 3rd.

Post-mortem examination.—The aorta was very atheromatous, and just above the diaphragm it was slightly bulged and contained a thin layer of ante-mortem clot. On cutting up the abdominal aorta the lower part for about 1½ inches was found to be entirely closed by a tough ante-mortem adherent clot, which commenced about 1½ inches above the bifurcation and extended to just above the orifice of the renal arteries—the vessel above this point being somewhat dilated and filled with a softening ante-mortem clot. Just below the orifice of the superior mesenteric artery and on the left side of the aorta was a saccular aneurysm about the size of a Tangerine orange, which had an orifice about an inch in diameter, closely plugged by a mass of rough, ridged and laminated clot, which was projecting slightly into the calibre of the vessel. The wall of the sac was very thin, and it had produced a good deal of erosion of the bodies of the first and second lumbar vertebræ. The superior mesenteric artery was plugged by a small rounded mass of ante-mortem clot which had blocked and become adherent to its orifice. The gastric, splenic, and hepatic vessels were quite free. The inferior mesenteric artery was also obstructed. The diaphragmatic arteries were large, but the epigastric and spermatic were not unusually large.

Embolism of the branches of the abdominal aorta is a rare occurrence. Embolism of the gastric arteries may give rise to hæmatemesis; of the splenic artery to pain and tenderness over the spleen; of the renal arteries to pain in the loins and hæmaturia; of the superior or inferior mesenteric arteries to sudden severe abdominal pain, abdominal distension, vomiting, hæmorrhage from the rectum, and collapse, followed, as a rule, by death in a few days.

Cause of Death.—Emaciation, increasing weakness, and extreme prostration occur as a result of the constant wearing pain and intense suffering, death from exhaustion and syncope following. In about half the cases death occurs suddenly from rupture of the sac into the peritoneal or pleural cavities. When the sac ruptures into the retro-peritoneal tissues the escape of blood is much slower, and at first a sense of relief from the pain may be experienced; but anæmia follows, and the patient probably succumbs to another or more extensive hæmorrhage. The indications of rupture having taken place are sudden collapse, intense pain, restlessness, pallor, profuse clammy perspiration, a rapid thready pulse, and a sudden subsidence of the tumour if the sac has ruptured into the pleural or peritoneal cavities.

Physical Signs.—The most characteristic signs of this disease which may be obtained by a physical examination are—a tumour with expansile pulsation, occasionally a thrill, and more frequently a systolic bruit. In addition there are the signs produced as a result of pressure on adjacent structures, and by the extravasation of blood into the retro-peritoneal tissues.

Case to illustrate the typical physical signs of aneurysm of the abdominal aorta.—A yeoman signaller in the navy æt. 24, came to my out-patients' on September 3rd, 1900, complaining of pain in his back. He had been five years in the navy, and had always been smart and active.

Digitized by Google

During the last two or three years his work had been mainly clerical. He denied ever having suffered from syphilis. When ten years of age he had a fall, which was followed by a swelling in his groin. He first noticed pain in his back about twelve months before admission, in consequence of which he had to stop any form of violent exertion, and about six months afterwards, when at Cardiff, he was seized with severe pain in his back, and had to go to bed. Three months later he was admitted into the Barracks Hospital, and he stated he was considered by the doctor to be a malingerer. The pain, which was always increased by walking exercise, had been continuous, dull and aching in character, and in consequence he had not been able to sleep. He had lost 40 lbs. in weight. For three or four months he had noticed a swelling in his abdomen.

He appeared to be fairly strong, but was thin and anæmic. A pulsating tumour was noticeable midway between the ensiform cartilage and the umbilicus, slightly to the left of the median line of the abdomen; it measured 3 inches by 2½ inches. The pulsation was distinctly expansile, and a systolic murmur was audible over the tumour. There was no evidence of pressure on the spinal cord. A diagnosis of aneurysm of the abdominal aorta was made, and he was admitted under the care of Dr. Hale White, who confirmed this opinion. On the 20th the right kidney was distinctly palpable, and this was thought to be due to its displacement by the aneurysm. On October 11th he was suffering from intense pain, which he referred to the middle of his back, and on the following day a large mass was noticed to have suddenly appeared in the right Scarpa's triangle, which was considered to be blood effused as a result of the rupture of the aneurysm. He died on the following day.

Post-mortem examination.—There was an aneurysm of the abdominal aorta measuring 7 by 5 cm., situated between the orifice of the cœliac axis and the spermatic arteries, the renal arteries coming off from the lateral wall of the sac. The inferior vena cava was lying stretched over the right side of the anterior surface of the aneurysm, and was firmly adherent to it. There were also adhesions fixing the sac to the pancreas and the third portion of the duodenum. The sac was full of recent blood-clot, and there was no sign of ante-mortem thrombosis having occurred. The aorta above and below the aneurysm appeared to be perfectly normal. A rupture had taken place on the right margin of the posterior border of the aneurysm, and the blood had been poured out into the substance of the right quadratus lumborum and psoas muscles, forming a tumour of considerable size. The blood had tracked downwards in the substance of the psoas muscle below Poupart's ligament into the right thigh. There was no blood lying free in the peritoneal cavity. The right kidney was pushed forwards and lying on the anterior surface of the mass formed by the extravasated blood. The anterior portions of the bodies of the first and second lumbar vertebræ were eroded by the aneurysm, the cartilages being intact.

There were no signs of syphilis found in the other organs of the body.

The position of the tumour varies, being dependent in the case of saccular aneurysm on the part of the aorta from which it rises, and in the case of diffuse aneurysm on the direction in which the blood extravasates after retro-peritoneal rupture has occurred.

The tumour may be of sufficient size to cause a local bulging of the abdominal parietes. The prominence thus caused occurs most frequently in the epigastric region, and is then due to the sac arising from the aorta in the immediate neighbourhood of the cœliac axis. When the sac increases in size to the left the bulging may be noticed in the left hypochondrium or left flank, and may even cause a local protrusion of the lower ribs. If the sac enlarges in the posterior direction a tumour may arise in the lumbar region and back, and if to the right in the right hypochondrium or loin. When the aneurysm is situated high up and just below the diaphragm, no definite tumour or bulging is, as a rule, apparent. If the sac arises from the aorta below the cœliac axis, the tumour is in the umbilical region.

When, in cases of diffuse aneurysm, the blood has burrowed into the quadratus and psoas muscles, the tumour may simulate a renal enlargement, and if the effusion extends in the sheath of the psoas below Poupart's ligament a tumour may appear in the groin and upper part of the thigh, which may be mistaken for a psoas abscess.

Digitized by Google

When the aneurysmal sac is sufficiently large to cause a local bulging of the abdominal wall, a close inspection of this area will show pulsation, which may be slight and almost imperceptible, or forcible and very marked. Pulsation is most evident in a saccular aneurysm which contains little or no thrombus. When the sac is filled with adherent clot, or if the aneurysm be diffuse, the pulsation is very slight. Pulsation may also be noticed even when the sac is not sufficiently large to cause any prominence of the abdominal wall.

When blood is extravasated into the anterior abdominal wall ecchymoses may appear, and in one case which I have already mentioned, in which blood was effused into the spermatic cord, the scrotum on the same side became much ecchymosed.

Œdema and dilatation of the superficial veins of the leg and lower part of the abdomen may be found in cases in which there is compression of the inferior vena cava; but these signs are exceptional unless the aneurysm is of the arterio-venous variety, when the œdema and the dilatation of the veins become extremely well marked.

On palpating the abdomen a pulsating tumour may be felt, and the pulsation determined to be distinctly expansile. If the patient be placed in the knee-elbow position, the pulsation does not disappear as it does when it happens to be due to a tumour lying over the front of a healthy aorta. Dr. Osler states that occasionally the pulsation is double when the sac is large and in contact with the pericardium.

A saccular aneurysm feels globular, smooth, regular, and elastic, and it may be compressible, tender, and even painful. When filled with adherent or organised thrombus it is hard, firm, non-compressible, and non-expansile, and if the opening into the aorta is blocked there is little or no pulsation.

A diffuse aneurysm may give rise to a tumour in the loin which may simulate a renal enlargement, and pulsation may be present or entirely absent. In some cases the kidney can be felt lying in front of the tumour.

A tumour due to an aneurysm of the abdominal aorta is not, as a rule, altered in position by the respiratory movements, nor is it at all moveable unless it arises at the orifice of one of the large branches, and the sac is formed partly by a distension of the aorta, and partly by a dilatation of that particular branch itself. Occasionally, but very rarely, a pulsating tumour may be detected in the position in which a psoas abscess can usually be felt.

The pulsation in the femoral arteries may be unequal, and pressure on the tumour may cause a distinct increase of tension in these arteries; whereas if the pressure is suddenly relaxed, a fall of tension and intermittency of the femoral pulse may be noticed for a few seconds.

A thrill can be felt in a few of the cases on palpating the tumour, the rhythm being systolic or slightly post-systolic, but it must not be relied on as an important diagnostic sign.

When an aneurysm is large, or has become diffuse, evidence of the displacement of certain viscera may be forthcoming. I have already mentioned displacement of the kidney downwards and forwards; the heart may be pushed upwards and outwards, and the liver downwards.

When the sac of the aneurysm is small or moderate in size it may be covered by the stomach, colon, or small intestines, and the percussion note obtained is resonant or subresonant. If the tumour causes a projection in the loin the percussion note is dull.

A murmur can be detected on auscultating the tumour in about 50 per cent. of the cases, and is usually best heard over the most prominent part of the aneurysm. It varies in intensity and character, in some cases being very loud, in others very faint; it may be soft, blowing, or musical; it is usually systolic, but occasionally is found to be post-systolic. It may be altered in character by a change of posture, or by a variation in the degree of pressure exerted by the stethoscope, but it is audible when the stethoscope is applied quite lightly to the abdominal wall. Dr. Osler states that a diastolic murmur, soft in quality, may occasionally be detected. If the aneurysm is diffuse, or if a saccular aneurysm is filled with adherent clot, the murmur may be absent.

In the case of arterio-venous aneurysm involving the aorta just above the bifurcation and the lower part of the inferior vena cava, which I have already described to you, a loud systolic bruit was heard along the course of the aorta, all over the abdomen, and the outer side and back of the thighs.

Digitized by Google

If the cardiac end of the stomach or the lower end of the œsophagus is compressed by the aneurysm, the abdomen becomes flat and retracted, and the patient rapidly emaciates. If the duodenum or pylorus be obstructed by pressure, or kinked by displacement, the stomach becomes dilated, and the physical signs of this condition are present.

If the bodies of the vertebræ are destroyed, and the spinal cord is compressed, weakness of the legs, loss of sensation, a band of hyperæsthesia around the body at the level of the lesion, exaggerated knee-jerks, extensor plantar responses, involuntary passage of urine and fæces are the result.

When blood has extravasated from an aneurysm into the retro-peritoneal tissues, and pushed forward the kidney and stretched the ureter, the testicle on the same side may become swollen and tender.

When rupture has occurred into the peritoneal or pleural cavities the tumour and the pulsation may disappear, and the signs of a large pleural or peritoneal effusion may suddenly become apparent.

Aneurysm of the abdominal aorta; rupture into the peritoneal cavity.—An omnibus conductor æt. 27, was admitted on June 30th, 1858, under the care of Dr. Wilks. He was of irregular habits, and had suffered from syphilis. Three months before admission he began to experience a sensation of beating in his abdomen, and he suffered from pain in his back and sides. Three weeks before coming to the hospital he noticed a tumour in his abdomen.

On admission he was pale and thin, and complained of pain in his back and across his abdomen. A projecting pulsating tumour was seen and felt a little to the right of the ensiform cartilage. A systolic bruit was heard over the front of the tumour, and behind there was a slight continuous murmur. He died suddenly on July 11th, and when examined the tumour was found to have subsided.

Post-mortem examination.—The peritoneal cavity contained several pounds of blood-clot. An aneurysm about the size of a duck's egg was found projecting above the lesser curvature of the stomach beneath the edge of the liver. The upper part of it had burst. It had formed at the origin of the cœliac axis, but had not involved that vessel, for the opening was at the side of the aorta. The arteries came off from the right side, and on the left was the aneurysm, which extended both backwards and forwards, and in this way the bodies of the two vertebræ were partly absorbed, at the same time that a tumour was formed anteriorly. The vessel above and below was healthy.

AN ATLAS OF HUMAN ANATOMY, for Students and Physicians. By CARL TOLDT, M.D., Professor of Anatomy in the University of Vienna; assisted by ALOIS DALLA ROSA, M.D. Translated from the third German edition and adapted to English and American and International terminology by M. EDEN PAUL, M.D.Brux., M.R.C.S., L.R.C.P. First Section: (A) The Regions of the Human Body. (B) Osteology (Figs. 1—377, and index). (Rebman, Ltd., 129, Shaftesbury Av., London.)

Dr. PAUL, who has translated this first section of Toldt's 'Atlas of Human Anatomy,' very truly remarks in his preface that even if no new anatomical data are likely to be forthcoming, there is yet scope for originality in the method of presentation of those data of which the science of human anatomy now consist; and we agree with the translator that originality of this kind is exhibited in a very high degree by the author of this work. In the many useful English manuals of human anatomy the illustrations, even when numerous, as they are often, and when good, as they are occasionally, form a mere supplement—usually a very imperfect supplement—to the text. This is the opinion of Dr. Paul, and many will be found to admit its truth. It must be, however, freely allowed that among English works an accurate pictorial representation of all the data of human anatomy, carefully drawn to scale from actual specimens, and arranged suitably for systematic study, has hitherto been lacking. The translator has in his preface so accurately put forward the true value of this work of Toldt's that we cannot refrain from further quoting his views. He says that whilst a true knowledge of anatomy—the knowledge that will through life supply the needs of the physician and the surgeon in their practical work—can be obtained only in the dissecting room, the student's labours with scalpel and forceps must be preceded and supplemented by systematic private study. For this purpose the textual descriptive treatise is not alone sufficient. Both in his work preparatory to dissection and in his revision of his anatomical knowledge subsequent to dissection, the energy of the student will be enormously economised if he has at hand a graphic representation of every structure named and described in his systematic treatise. An increased use of the visual or graphic method, both in the acquirement and in the revivification of knowledge, is a feature of the age in all educational departments; but this English translation of Toldt's 'Anatomy' is, as far as the English-speaking races are concerned, the first adequate application of the method to the study of human anatomy. The publication of this work is arranged in six sections, and it is intended to issue a section every month till the atlas is completed. We shall be curious to see if the remaining six sections of this work reach the high standard of excellence attained in the volume before us.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 578. **WEDNESDAY, NOVEMBER 25, 1903.** **Vol. XXIII. No. 6.**

CONTENTS.

PAGE

*A Lecture on the Modification of Cow's Milk for Infant Feeding. By GEORGE F. STILL, M.A., M.D., F.R.C.P. 81

*Two Clinical Lectures on Aneurysm of the Abdominal Aorta. By J. H. BRYANT, M.D., F.R.C.P. Lecture II ... 89

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A LECTURE

ON

THE MODIFICATION OF COW'S MILK FOR INFANT-FEEDING.

Delivered at the Hospital for Sick Children, Great Ormond Street.

By GEORGE F. STILL, M.A., M.D., F.R.C.P.,

Assistant Physician to the Hospital; Assistant Physician for Diseases of Children, King's College Hospital.

GENTLEMEN,—I suppose that most of us when we first entered upon the practice of our profession cherished a faith that, failing the mother's milk, the only proper food for an infant was cow's milk, and that it was the simplest thing in the world to feed an infant successfully if only this golden rule were remembered. A very little experience, alas! and any such belief in the simplicity of infant-feeding has been rudely shaken; often, indeed, if cow's milk is ordered at all, it is ordered with fear and trembling, and on the first sign of its disagreeing recourse is had to some patent food or condensed milk.

Far be it from me to pass any sweeping condemnation on such methods of feeding; it is the abuse, not the use of these foods and condensed milks which is so disastrous; used with an intelligent appreciation of their composition, and of the ages and conditions for which they are suitable, they undoubtedly have their value. But none the less it is true as a general statement that there is not a single patent food or condensed milk in the market which can adequately replace fresh cow's milk in the feeding of an infant who is deprived of the mother's milk; and moreover, that a vast amount of infantile suffering and misery, and no small sacrifice of infant life, are due solely to the indiscriminate feeding of infants with these artificial preparations.

Even when the unsatisfactory result of such

Digitized by Google

resources is evident in gastro-intestinal trouble of one kind or another, or in failure to gain weight as the infant should, any proposal to give cow's milk is commonly met with the objection, "It is no use ordering cow's milk; we have tried that, and the baby can't take it." In such cases the question always arises in my mind, "Has sufficient care been taken in the modification of the milk?" And too often one finds that a mixture has been used which differs so widely from human milk that it is little wonder if the infant fails to thrive upon it. Judging from my own experience, I am inclined to think that there is a certain number of infants who cannot take cow's milk, modify it as you will; but I am convinced that this number is *extremely* small, and that the vast majority of infants will thrive excellently on cow's milk, provided this has been properly modified.

The first and absolute essential for the intelligent modification of cow's milk for infant-feeding is a knowledge—I mean a practical working knowledge—of the exact average composition of human milk and cow's milk. Undoubtedly mere rule of thumb will serve for the feeding of many a vigorous infant; but if our sole idea of modification of milk is to dilute it once, twice, or thrice, and throw in "a little sugar" and, perchance, "some cream," trusting to luck or to the mother, or, worse still, to the monthly or domestic nurse, to adjust the proportions, we shall certainly have many failures in our efforts to feed even healthy infants, and many more in the case of weakly infants or those whose digestion has already been impaired by disease or unsuitable diet. It is especially these more difficult cases—and are they not common enough in everyone's practice?—in which accurate and intelligent modification of cow's milk is all-important.

Now, as in this lecture I shall have to refer again and again to the percentage composition of milk and of various modifications of milk, let me say at the outset that such percentages are only a convenient method of calculating approximately the composition of the food; and for clinical purposes a strictly exact percentage of any one constituent is neither necessary nor practicable. We have heard a great deal recently about "percentage-feeding," as if it were some great discovery. Let me remind you that there is nothing new in "percentage-feeding;" like Monsieur Jourdain with his "prose," we have used it all our medical lives, whether we knew it or not; the only innovation—and a very important one—is that the medical man should *know* approximately what percentage he is giving. It makes no difference whatever, whether you prescribe equal parts of milk and water, or prefer to order casein, 1·625 per cent.; lactalbumen, 0·375 per cent.; fat, 1·75 per cent., etc. (assuming the average composition of cow's milk to be as stated below); but it does make a very great difference whether you realise that the difficulty in digesting cow's milk lies in the large proportion of casein * which it contains, and whether you understand how to get over this difficulty without reducing the other important constituents of milk to a harmful degree, or falling into the not less disastrous mistake of making the food too rich in cream or sugar.

I put before you now the average composition of human milk and cow's milk. For convenience' sake I shall leave the salts out of consideration; they undoubtedly have their importance, but we know so little about them, and they probably play such a comparatively insignificant part in nutrition, that it is hardly worth while to burden our memories with their percentage. The proportion of water can also be left unmentioned; it forms, of course, the residue of the percentage.

Human milk.	Cow's milk.
Proteid, 2 per cent. { Caseinogen, 0·6 per cent. Lactalbumen, 1·4 per cent.	Proteid, 4 per cent. { Caseinogen, 3·25 per cent. Lactalbumen, 0·75 per cent.
Fat, 3·5 per cent.	Fat, 3·5 per cent.
Sugar, 7 per cent.	Sugar, 4 per cent.

As I have already said, the one great obstacle to the digestion of cow's milk is the large quantity of curd which is formed when it enters the stomach, and this is due to the heavy percentage of caseinogen which it contains. This caseinogen forms a curd when mixed either with acid or with rennet, whereas the remaining proteid, lactalbumen, is only coagulated by heating above 160° F.

Lactalbumen is so easy of digestion that so far as this portion of the proteid is concerned no

* The terms casein and caseinogen, strictly used, refer respectively to the formed curd and the potential curd, *i. e.* the curd-forming proteid before it is coagulated; for brevity's sake the term casein has been used sometimes in this lecture where caseinogen would have been more strictly accurate. [G. F. S.]

Digitized by Google

dilution of cow's milk is necessary; the only object of dilution is the reduction of the caseinogen to a proportion which the infant can digest.

If human milk is to be taken as the measure of a healthy infant's power of digestion, it is clear that in order to reduce the proportion of caseinogen in cow's milk to this standard, considerable dilution is necessary. Fortunately nature has provided even the youngest infant with some range of accommodation in its powers of digestion, and many an infant will digest a larger proportion of caseinogen in cow's milk than the average composition of mother's milk might suggest.

But, notwithstanding this power of accommodation, I think I shall be correct in saying that a large majority of the failures in infant-feeding with cow's milk are due to insufficient dilution.

For an infant two or three weeks old it is a common practice to order a mixture of one part of milk with two parts of water—that is, 1 in 3. This, of course, means division of each percentage by 3; and a comparison with human milk shows how widely such a mixture differs therefrom.

Human milk.	Cow's milk and water, 1 in 3.
Proteid, 2 per cent. (Caseinogen, 0·6 per cent.; Lactalbumen, 1·4 per cent.)	Pro teid, 1·3 per cent. (Caseinogen, 1·08 per cent.; Lactalbumen, 0·25 per cent.)
Fat, 3·5 per cent.	Fat, 1·16 per cent.
Sugar, 7 per cent.	Sugar, 1·3 per cent.

The caseinogen is still nearly double the proportion which occurs in human milk. (I am not at present considering the other constituents; my object now is only to show you how far the ordinary dilution fails in its sole purpose—namely, the reduction of the caseinogen, the curd-forming proteid.)

Even if three parts of water are used with one of milk, there is still a slight excess of caseinogen, as can be seen from the following comparison (the percentages will, of course, be divided by 4):

Human milk.	Cow's milk and water, 1 in 4.
Proteid, 2 per cent. (Caseinogen, 0·6 per cent.; Lactalbumen, 1·4 per cent.)	Proteid, 1 per cent. (Caseinogen, 0·81 per cent.; Lactalbumen, 0·18 per cent.)
Fat, 3·5 per cent.	Fat, 0·87 per cent.
Sugar, 7 per cent.	Sugar, 1 per cent.

It is evident that in order to reduce the curd-forming proteid to the same proportion as is present in the mother's milk, four parts of diluent must be added to one part of milk (1 in 5). The percentages will thus be divided by 5, and the result will be as shown here:

Human milk.	Cow's milk and water, 1 in 5.
Proteid, 2 per cent. (Caseinogen, 0·6 per cent.; Lactalbumen, 1·4 per cent.)	Proteid, 0·8 per cent. (Caseinogen, 0·65 per cent.; Lactalbumen, 0·15 per cent.)
Fat, 3·5 per cent.	Fat, 0·7 per cent.
Sugar, 7 per cent.	Sugar, 0·8 per cent.

But how often is milk diluted to this extent? Hardly ever, even during the first few weeks of the infant's life; and yet it is considered a matter of surprise—or at any rate an evidence of some peculiar weakness in the infant's digestion—that an infant suffers with colic or vomiting or other gastro-intestinal trouble, and fails to thrive when—say at eight weeks old—it is having milk diluted with only two parts of water or barley-water. Surely the wonder is that so many infants manage to digest their food in spite of the excess of curd which the usual dilution entails.

Now I do not for a moment wish to advocate any extreme dilution for the vigorous infant who can digest the larger proportion of curd—the more of this that the child can take and digest the better,—but I do wish to insist very strongly on the necessity for more accurate modification of cow's milk, if only in respect of the faulty excess of curd, before rejecting this food in favour of any of the far less satisfactory substitutes with which the market is flooded.

In a very large number of cases where milk-feeding is tried and found wanting, the failure is due simply to insufficient dilution. Even at the age of five or six months some infants cannot digest a mixture of equal parts of cow's milk and water (caseinogen, 1·625 per cent.); and when it is remembered that the proportion of curd in mother's milk scarcely increases at all during the later months of lactation, we might expect that even at such an age it may be necessary to dilute cow's milk, if not to so great an extent as in the earlier months, at any rate much further than is usually done.

So far I have considered only the curd-forming proteid in cow's milk, but obviously any such dilution as I have mentioned involves serious alteration in the proportion of the other constituents also; and no doubt it is this fact which

Digitized by Google

accounts for the common practice of insufficient dilution.

The proportion of lactalbumen, which at the outset is little more than half that in human milk, becomes still more deficient if the ordinary diluents —such as water, barley-water, lime-water, etc.—are used.

On this point I shall have more to say later; here I will only point out that whilst it is the proportion of caseinogen, not the proportion of total proteids, which has to be corrected by dilution, it is nevertheless important that the resulting total of caseinogen *plus* lactalbumen should not be far below that present in human milk. The right course in determining the degree of dilution in any given case is to determine it solely with reference to the proportion of curd-forming proteid which the infant can digest, and then in one way or another to rectify as far as possible the deficiency of lactalbumen, fat, and sugar. It is the curd-forming part of the proteids—the caseinogen alone,—not the lactalbumen, which has to be considered in deciding what degree of dilution is necessary.

The deficiency which perhaps gives most trouble in the dilution of milk is the low fat percentage. You will see that when four parts of diluent are added to one part of milk to correct the proportion of caseinogen the percentage of fat becomes 0·7, and this, of course, is altogether too little; the proportion of fat should not be below 3 per cent., as a general rule, and many infants will thrive best with a mixture containing 3·5 per cent., or even 4 per cent. of fat. A low percentage of fat—for instance, 2 per cent. or less in the food—is almost certainly one of the commonest causes of rickets.

But the fact that dilution which is sufficient to correct the excess of curd-forming proteid involves at the same time diminution of the proportion of fat, is no justification for giving the milk only slightly diluted. I would remind you that with any of the ordinary dilutions there is necessarily some deficiency greater or less in the proportion of fat, unless cream be added or the fault corrected in some other way; and if such correction must be made—as it certainly must if the best results are to be obtained—it is just as easy to correct a large deficiency as a small one.

If we had a standardised cream, it would be perfectly simple to correct the percentage of fat in our diluted mixture by addition of the required proportion of cream. Such a cream is, I think, much needed; at the present time, so far as I know, there is only one dairy in London which regularly supplies a cream containing a known percentage of fat, warranted to contain a constant and declared percentage. But in the absence of such cream what is to be done? There are several ways of getting out of the difficulty. It is sometimes convenient to use "top-milk," by which is meant the upper portion of milk which has been allowed to stand in a vessel until the cream (*i. e.* the fat) has risen to the top; if this upper part be used for dilution instead of the lower, it has the advantage of containing the additional fat which has risen from the lower part, whilst the proportion of proteids is scarcely altered, so that the result of subsequent dilution is to diminish the proteid without causing as great a deficiency of fat as occurs when ordinary milk is diluted.

With average London milk (*i. e.* milk which contained 3·2 to 3·5 per cent. of fat) which had been allowed to stand three hours, I obtained the following results:

Quantity of top-milk taken from each quart of milk.	Proportion of fat.
One pint	5·5 per cent.
Fifteen ounces	6·5 „
Ten ounces	8·0 „
Five ounces	11·0 „

But such a method is, I think, unsatisfactory; the resulting percentages of fat are subject to considerable variation, and as this variation occurs in a comparatively small percentage, it makes the use of such cream unreliable where accurate modification of milk is necessary. The richer gravity cream —that is, cream made by allowing milk to stand several hours, and then skimming off the cream which has risen without taking with it any of the milk below the level of the cream—is open not only to the objection of variability of richness, but also to a more serious one—namely, that its preparation involves prolonged standing of the milk, which at any time, but especially in warm weather, is accompanied with obvious risk of chemical change from bacterial contamination. I have known cream to be prepared in this way by allowing ordinary London milk to stand as long as twelve hours after it had been delivered to the retail purchaser; and as such milk was probably at least twelve hours stale when it was delivered, and the cream made

Digitized by Google

from it would be used for infant-feeding several hours after its preparation was completed, the practical risk of bacterial contamination must be a very real one, however much it may be possible in theory to reduce it by special precautions.

In the country, where milk is available for home use almost directly it is drawn from the cow, this objection is less serious; but even so it cannot be desirable that milk should be left standing fifteen to seventeen hours, as it sometimes is, for the cream to rise. Where, however, only such cream is available, it may be useful to know that a quart of milk allowed to stand twelve to fifteen hours at the ordinary temperature of a cool dairy will yield a cream containing 17 to 25 per cent. of fat; and, as a working hypothesis, it is safe to assume that simple gravity cream (not clotted cream) contains 24 per cent. of fat; some samples will contain less, very few will contain more. Far better than any such methods is the use of centrifugal cream, such as is supplied by any of the large dairy companies in London and is commonly sold in the shops of London and other large towns. In the preparation of such cream there is no standing of the milk for many hours; the cream can be separated from the milk in a few minutes by a centrifugal machine, and with such a machine working at a fairly constant rate the cream made at any particular dairy shows only slight variations in strength.

Out of twenty-three samples bought from twelve shops in various parts of London, sixteen showed a proportion of fat between 40 and 50 per cent.; only three contained less than 40 per cent. (36, 36·8, and 38·4 per cent. respectively); five showed proportions varying from 50 to 54 per cent.; the average fat percentage of the whole twenty-three samples was 46·3.

Now the addition of fat which is required in any mixture for infant-feeding is practically never more than 3 per cent., and much more often only 2 or 2·5 per cent. is required. In order to obtain this with a cream containing 40 to 50 per cent. of fat, a dilution of 1 in 16 at least is necessary; and it is evident that an error even of 16 per cent. in the assumed fat percentage of the cream would involve only an error of 1 per cent. in the fat of the mixture ordered. For practical purposes 48 per cent. may be taken as the average proportion of fat in centrifugal cream—this is, I think, a safer average than 46·3, which was obtained only when the few exceptionally low percentages were included,—and on this assumption the error either way cannot be more than 10 per cent. at the utmost, and will very rarely be more than 6 per cent. in either direction; so that if we ordered, as is commonly necessary, a dilution of 1 in 24—that is, 2 per cent. of fat (the cream must be assumed to contain 48 per cent. of fat),—the utmost possible error is less than 0·5 per cent. in either direction, and it will rarely be as much as 0·25 per cent.

It is therefore a perfectly simple matter to correct the fat deficiency in any mixture. For example, suppose the infant is to have one part of milk with three of water, this will reduce the fat in the milk from 3·5 to 0·8 per cent. approximately; it is necessary to add sufficient cream to raise the percentage from 0·8 to 3—3·5 per cent. The addition of one drachm of cream (assumed to contain 48 per cent. fat) to 2½ ounces of the mixture, *i. e.* 1 to 20, will be the addition of 2·4 per cent. fat approximately, raising the total fat percentage of the mixture to 3·2 per cent.

A further advantage of this high-percentage centrifugal cream is the very small proportion which it is necessary to add to any milk mixture; this makes it possible to neglect all the other constituents of the cream, except the fat, in calculating the resulting percentages in the mixture. With any weaker cream of which a larger quantity must be added, it becomes necessary to consider the proteid and sugar in the cream as well as in the milk, and this makes the matter somewhat complicated.

So much for the correction of the fat deficiency. I come now to the correction of the deficiency of sugar, which is easy enough.

But here, again, I would have you believe that Nature is not the hard task-master which some writers would make her, and it is not necessary to adhere rigidly to any exact percentage. Some infants thrive excellently on 4 to 5 per cent. of sugar, while others will do equally well on 8 to 10 per cent. But none the less I think that Nature has shown us the most generally suitable proportion in the milk which she has provided —namely, the mother's milk, which contains 6 to 7 per cent. of sugar; and some infants are troubled with flatulence or colic or diarrhœa directly this proportion is exceeded even by as much as 1 per cent. It is evident that in using

Digitized by Google

cow's milk for infant-feeding, even if it were used undiluted, some addition of sugar would be desirable, for it contains only 4 to 5 per cent. of milk sugar. Dilution involves reduction of this proportion; for example, if milk be diluted with three parts of water the percentage of sugar will be only 1 per cent.

Elaborate methods have been devised for adding the exact quantity of sugar necessary to yield a proportion of 7 per cent., and with this object some writers have recommended the use of a solution of sugar as a diluent for cow's milk. This seems to me far less satisfactory than the use of the dry sugar itself; the solution is much less convenient for storage, and, what is much more important, it adds an unnecessary risk to infant-feeding by affording an excellent medium for the growth of micro-organisms whilst it is waiting to be used. The argument in favour of it is the facility it offers for exactly modifying the percentage of sugar when a standardised solution is at hand, and this, of course, is easily prepared.

But I would venture to object that in the first place no such extreme exactitude is necessary; and in the second, a quite sufficiently exact percentage is obtainable by simpler methods.

A level teaspoonful (using a teaspoon which holds two drachms of water, *i. e.* rather a large domestic teaspoon) of milk sugar in three ounces of any fluid gives a 5 per cent. solution of sugar. Therefore if this quantity be added to a three-ounce feed of any milk mixture, 5 per cent. of sugar has been added; and as the original proportion of sugar in the mixture is readily calculated by simple division according to the number of times the milk has been diluted, the total percentage, after addition of the milk sugar, is known without any difficulty. Similarly a lump of ordinary white cane sugar half an inch square in three ounces of fluid gives a 5 per cent. solution.

With these two data alone it is easy to regulate the percentage of sugar accurately enough for the feeding of an infant with the most delicate digestion. The quantity of sugar which must be added to a feed of any amount, or of any proportions, can easily be calculated, and also easily measured with sufficient accuracy to ensure a proportion of 5 to 7 per cent. of sugar. As a matter of fact, with any of the ordinary milk mixtures made with such diluents as water, barley-water, or lime-water, the addition of 5 per cent. of sugar will give a total percentage which is quite near enough to Nature's average for practical purposes. For instance, if the milk has been diluted with two parts of water, the sugar will have been reduced to 1·3 per cent.; and the addition of sugar in the proportion of one teaspoonful of milk sugar, or one lump half an inch square of cane sugar, to three ounces of the milk mixture, will raise the total sugar percentage to 6·3; while if two parts of milk to one of water are being given, the mixture will contain 2·6 per cent. of sugar, and addition of sugar in the proportion suggested will raise the percentage to 7·6. By making the added teaspoonful of sugar slightly less full, or reducing the size of the lump of sugar added, it is easy to keep the resulting percentage within the 7 per cent. limit, if desired.

The choice between milk sugar and cane sugar is not, I think, one of very great importance, but I prefer the former if expense is no difficulty, for it has seemed to me less liable to cause flatulence. Now let me put before you the result of such modification as I have so far considered, taking as a very simple example a three-ounce mixture in which one part of milk has been mixed with three parts of water. (The cream, as I have said, may be considered merely as an addition of fat; its fluid bulk and also the proteid and sugar constituents of cream may be left out of account, for they bear so minute a proportion to the whole mixture that they may well be neglected for the sake of convenience.)

Mixture.		Resulting percentages.	
Milk	6 drachms.	Casein	0·81 per cent.
Water	18 „	Lactalbumen	0·18 „
Cream (48 per cent. fat)	1½ „	Fat	3·87 „
Milk sugar	1 level teaspoonful.	Sugar	6·00 „

The obvious defect in such a mixture as compared with human milk is the low percentage of lactalbumen—a defect which is present to some extent in cow's milk, even before dilution, but which is made much more serious when any of the ordinary diluents (water, barley-water, lime-water) are used to dilute the milk.

And this brings me to the question of diluents. What diluent is best in the modification of milk? I venture to say there is one, and only one ideal diluent, and that is whey. Whey is, in fact, Nature's

Digitized by Google

diluent for milk, and milk may be regarded as whey containing in suspension curd-forming proteid and fat. When the curd is strained off it carries with it the greater part of the fat, and there is left a clear or slightly turbid fluid, the whey, which contains all the original constituents of milk except the caseinogen and fat (a varying proportion of the fat, but rarely more than 1 per cent., according to my own analyses, can be retained in the whey, if the curd is thoroughly broken up before straining); in particular the whey contains practically the same proportion of lactalbumen salts and sugar as was originally present in the milk.

If, therefore, whey be used as a diluent, however far the dilution be carried, in order to reduce the proportion of curd-forming proteid (caseinogen), there will be no dilution whatever of the lactalbumen.

This surely is a great gain. In the example which I have just put before you, the substitution of whey for water would raise the proportion of lactalbumen from 0·18 per cent. to 0·75 per cent. and the proportion of total proteids, therefore, from about 1 per cent. to 1·56 per cent.; and this increase in the proteids is obtained without increasing the difficulty of digestion.

The use of whey involves some care in its preparation, if the whey is to be of the greatest possible value. The rennet or curdling ferment which is used to prepare the whey must be destroyed by heat before the whey is used as a diluent; otherwise the ferment, being still active, will curdle the milk to which the whey is added; but if a temperature above 160° is used the lactalbumen is coagulated. To avoid this a temperature of 150° to 155° may be used, which destroys the ferment but does not coagulate the lactalbumen. This is, I think, desirable, but not absolutely necessary; the change in the lactalbumen, which results from heating even to boiling point, seems to interfere little if at all with its digestibility, and probably equally little with its nutritive value. Of course, if whey is used as a diluent it must be remembered that the percentage of sugar in the milk is not reduced at all, and therefore less addition is necessary to raise the 4 per cent. present to the required 6 to 7 per cent. The fat in the whey must also be considered. If the whey has been made without breaking up the curd at all, the amount of fat present (0·2 to 0·5 per cent.) is so small that it might almost be disregarded; but as some breaking up of the curd has usually taken place before the whey is strained off, it is generally more accurate, and certainly safer for practical purposes, to assume that the whey contains 1 per cent. of fat, and that therefore the resulting deficiency of fat when milk is diluted with whey is 1 per cent. less than it would be if water were used as diluent.

Now compare the following modification with that which I have just shown you:

Mixture.		Resulting percentages.	
Milk	6 drachms.	Casein	0·81 per cent.
Whey	18 „	Lactalbumen	0·75 „
Cream (48 per cent. fat)	1 „	Fat	3·87 „
Milk	½ level teaspoonful.	Sugar	6·50 „

Here the proportion of curd-forming proteid caseinogen has been reduced without reducing the proportion of lactalbumen; the mixture approximates more nearly to human milk in respect of its lactalbumen than is possible with any other diluent.

The preparation of whey, of course, entails a certain amount of trouble, and makes infant-feeding a little more expensive than when plain water or barley-water is used as diluent; but these disadvantages are, I think, altogether outweighed by its advantages, and are not sufficient to prevent even hospital out-patients using such whey dilution for several months, as I have found in my own hospital experience.

If whey cannot be obtained, barley-water has no doubt some advantage over plain water in rendering curd more flocculent, and perhaps in increasing to a very small degree the nutritive value of the mixture; but I have no great liking for its use in infant-feeding. I believe that I have several times seen slight degrees of rickets result from its prolonged use, and there is no doubt that it is not uncommonly the cause of flatulence and discomfort, and sometimes of looseness of the bowels. I have said nothing with regard to that very important point, the pasteurisation of milk for infant-feeding. I should like to have contrasted pasteurisation with sterilisation, a process which in my opinion is even more unnecessary and more injurious to the milk than is the common practice of

Digitized by Google

boiling for several minutes. This, however, and other points I must leave unconsidered, for I wish to say a few words in conclusion with reference to the "laboratory modification," and "percentage prescribing" of milk, of which we have heard so much within the past few years. How far is any such exact modification practicable? A very interesting paper has recently been written by Dr. Wentworth, of Boston, U.S.A., showing that of twenty-six milk prescriptions made up at a milk laboratory in that city, not a single one corresponded exactly with what was ordered; there was as much as 0·2 per cent. variation in the proteids, and as much as 1 per cent. error in the fat and in the sugar. I have made a few control analyses of the same kind myself, and have found small variations (as much as 0·5 per cent. in the fat and rather less in the proteids) from the formula ordered; but I confess I am more inclined to wonder at the degree of accuracy which was attained than to criticise these slight deviations. At the same time these observations show that even at a special laboratory the percentages obtained are, if more accurate than those obtained with home modification, still only approximate; and to expend scrupulous care over the second place of decimals—as I have seen done—in writing a "milk prescription" is either to humbug ourselves or our patients.

But is any such strict accuracy either necessary or desirable? Surely not, if Nature is to be our guide. Human milk varies not only with the health of the mother and her diet, but even with the time of day and with the portion of breast milk taken. I have found a difference of 1 per cent. in the fat in two samples of a woman's milk taken at 11 a.m. and 11 p.m. respectively, and I have repeatedly found the morning milk of women to be richer in fat than the evening milk. In the milk first drawn from the breast I have found as little as 0·4 per cent. of fat when the middle milk at the same feed contained 3·2 per cent. of fat, so that obviously the infant will get a food varying in its average composition according to the completeness with which it empties the breast. A rigidly fixed percentage of each constituent is not only unnecessary, but is also unnatural.

Nevertheless it is clear that there is a certain range of variation in the proportion of each constituent which is permissible for any particular infant, and this range has no very wide limits. The value of a knowledge of the percentage composition of milk, and the methods by which the percentage of each constituent can be regulated approximately, seems to me to lie in its enabling us to determine with some degree of accuracy what these limits are; and when we have determined them, to regulate the proportion of each constituent so that it falls within this range. Where expense is no object it is a very great convenience to have milk mixtures supplied with the proportions already regulated according to the medical man's instructions; but let it be realised that this regulation of percentages is carried out in exactly the same way as is done in the home modification of milk, and that any medical man who possesses a working knowledge of the average composition of milk, cream, and whey (or, still better, can obtain analyses of the particular milk and cream which are to be used), can regulate the percentages of proteids, fat, and sugar with sufficient accuracy for any practical need of infant-feeding.

Veratrum Viride in Health and in Eclampsia.—Chidichimo's researches on dogs and other laboratory animals indicate that veratrum viride has a marked depressant action in physiological conditions. It rapidly reduces the temperature, and this reduction continues for hours, while about thirty hours are required before the drug is eliminated from the organism in the case of rabbits and dogs. The facts observed indicate also that it acts mainly as a toxin on the muscles. On the whole, his experience would impel him to proclaim that veratrum viride should be banished from the treatment of puerperal eclampsia if it were not for the possibility that the organism in the stress of eclampsia may react differently to the drug than might be expected from its physiological action on animals. Two subcutaneous injections of 1 cubic centimetre of the Italian fluid extract of veratrum viride were given by Pinzani, with a half-hour interval, after forceps extraction of a dead fœtus. The same amount was given in a single dose to a second patient. The fœtus was extracted later with forceps, as it was seen to be succumbing. The same amount was injected in the third case, but with a longer interval. Improvement was marked in all after the injections, as also in six other cases treated in the same way and previously reported. The women all recovered, but the fœtus did not survive. The total amounts ranged from 1 to 4 cubic centimetres, never more than 3 cubic centimetres in one dose. The pulse is the guide; as this becomes less rapid the convulsions subside. —*Monthly Cyclopædia*, October, 1903.

Digitized by Google

TWO CLINICAL LECTURES
ON
ANEURYSM OF THE ABDOMINAL AORTA.

Delivered at Guy's Hospital, July 8th and 15th, 1903.

By J. H. BRYANT, M.D., F.R.C.P.,
Assistant Physician, Guy's Hospital.

LECTURE II.

GENTLEMEN,—To-day I shall discuss with you the diagnosis, prognosis, and treatment of aneurysm of the abdominal aorta.

DIAGNOSIS.—When a patient complains of severe pain in the abdomen and back, and on making a physical examination a deep-seated abdominal tumour with marked expansile pulsation and a systolic bruit is found, the presence of an abdominal aneurysm may be assumed, but there must be no question about the expansile character of the pulsation. In a large proportion of cases, however, all these cardinal signs are not present, and then a correct diagnosis becomes a matter of great difficulty, and may not be made until rupture of the sac occurs, or even a post-mortem examination is performed. That such difficulties actually arise is well exemplified by the fact that a correct conclusion during life as to the nature of the disease was arrived at in eighteen only out of the fifty-four cases on which this lecture is based, an analysis showing that an abdominal tumour was detected in thirty-one, pulsation in thirty-five, expansile pulsation in eight only, and a systolic murmur in twenty-six. Incorrect diagnoses of a variety of diseases were made, including malignant tumours lying in front of the aorta, renal calculus, lead colic, spinal caries, sarcoma of the kidney, nephritis, perinephritis, pneumothorax, pleuritic effusion, epithelioma of the œsophagus, malingering, chronic intestinal obstruction, etc.

A diagnosis of abdominal aneurysm is made much oftener when it is not present than an incorrect diagnosis when it is present, and the condition which most frequently leads to this mistake is pulsating abdominal aorta.

I take this opportunity to warn you against diagnosing an abdominal aneurysm on insufficient evidence. Do not be led away or biassed by the presence of abdominal pulsation, for excessive pulsation of the abdominal aorta is by no means uncommon. Examine the abdomen with great care, make your mind quite clear about the character of the pulsation, whether it is expansile or transmitted, and determine if a definite tumour is present or not. In thin subjects the bodies of the vertebræ may be readily palpated, especially if there happens to be any degree of lordosis, and be mistaken for an abdominal tumour, but a careful bimanual manipulation should enable you to determine the nature of such a pseudo-tumour.

In my experience, excessive pulsation of the abdominal aorta is much commoner in women than in men, and I can recall a large number of patients who have been sent to me as cases of abdominal aneurysm, who were suffering from this condition,—in fact, I have examined four such cases during the last fortnight. The more frequent occurrence of excessive pulsation of the abdominal aorta in women than in men, and the extreme rarity of aneurysm of the abdominal aorta in women, should make the physician hesitate before committing himself to a diagnosis of such a serious and fatal disease, especially in a female. Let me again emphasize this point by referring to the important and striking fact that only five out of the fifty-four cases were in women. Of these five, one was a child who had a commencing aneurysm at the end of the aorta, the result of embolism from an infective endocarditis,—so that in reality there were only four cases of aneurysm of the abdominal aorta in women out of fifty-four cases spread over a period of forty-seven years.

Women who suffer from pulsating abdominal aorta are usually anæmic, dyspeptic, and decidedly neurotic. They complain of abdominal pain, vomiting, and a sensation of throbbing in the abdomen. A careful physical examination may enable you to determine that there is no tumour, and that the vessel is of normal size. A systolic murmur is not present unless the stethoscope is pressed sufficiently hard against the abdominal wall to slightly alter the contour of the vessel and form a fluid vein. The abdominal wall is usually loose and flaccid, and the contained viscera are often displaced and moveable, especially the kidneys.

The following is a good illustrative case of this kind:

A lady twenty-seven years of age, who had borne

Digitized by Google

two healthy children, complained of abdominal pain, which was associated with a more or less constant sensation of throbbing, and was liable to rather severe exacerbations. Her father and her husband, both of whom were medical men, were feeling anxious on the score of abdominal aneurysm, and the lady herself was somewhat disturbed and alarmed at the possibility of her symptoms being due to such a serious condition. A physical examination revealed a loose and flaccid abdominal wall, movable kidneys, a slightly displaced and dilated stomach, and marked epigastric pulsation, but no tumour in the line of the aorta, which vessel could be distinctly felt to be of normal size. An assurance that her symptoms were due neither to an abdominal aneurysm nor to a cancer, the use of an abdominal support, in the form of a bandage, careful dieting, and a mixture containing nux vomica, bromide of soda, sal volatile, and compound infusion of gentian was followed by an almost immediate relief of her symptoms.

Pulsation in the epigastrium from other causes, viz. enlargement of the right ventricle, pulsating liver, and pulsating sarcomatous tumours may simulate aneurysm, but should not present much difficulty if a careful examination of the patient is made.

With regard to the pulsation that may be due to hypertrophy or dilatation of the heart, you should remember that it is extremely rare to find any increase in the size of the heart, either from hypertrophy or dilatation, as a direct result of aneurysm of any part of the aorta, with the exception of the intra-pericardial portion, which leads to aortic regurgitation, and so to hypertrophy and dilatation of the left ventricle. If, therefore, epigastric pulsation is associated with the signs of a considerable increase in the size of the heart, it is not likely to be the result of an aneurysm of the abdominal aorta. Hypertrophy and dilatation of the heart, however, must be distinguished from displacement of the heart, a condition which may be produced by a large aneurysm arising from the aorta just below the diaphragm.

Pulsation of the liver is associated with enlargement of that organ, and with signs of chronic valvular disease of the heart, or of chronic bronchitis and emphysema.

A rapidly growing vascular sarcoma of the left lobe of the liver may give rise to a tumour which pulsates, and over which a bruit may be heard. A careful physical examination would, however, enable you to determine its connection with the liver, which organ, in all probability, would be considerably enlarged.

I have seen a case of sarcoma of the mesenteric glands, giving rise to a tumour just above and to the left of the umbilicus, mistaken for an abdominal aneurysm, the correct diagnosis not being made until an explanatory laparotomy had been performed.

Malignant tumours lying over the front of the aorta, especially of the pyloric end of the stomach, pulsate on account of their close relationship to that vessel. The pulsation, however, is not expansile, but distinctly transmitted, and the tumour may be found to move downwards on inspiration, a condition which is rarely associated with aneurysm of the abdominal aorta. Another important distinguishing test is to examine the patient in the knee-elbow position, when the pulsation will disappear if it is due to a malignant tumour, but will not do so if the tumour is an aneurysm.

In some cases of aneurysm of the abdominal aorta a diagnosis of renal calculus or some pathological change associated with it, such as perinephritic abscess, has been made; and although it may seem strange to you that two diseases so very different from each other may be mistaken, a comparison of the symptoms under certain conditions may show many points of great similarity. When the sac arises from the aorta in such a position that as it enlarges it pushes forward the kidney and puts the ureter on the stretch, the pain which results may be sharp and paroxysmal in character, may shoot along the course of the ureter into the scrotum, and the testicle may not only become retracted, but be swollen, tender, and painful. The two following cases illustrate the difficulties which may arise.

Aneurysm of the abdominal aorta diagnosed renal calculus.—A shoemaker æt. 49, was admitted under the care of Dr. Habershon on November 18th, 1866, for pain in his left loin. When twenty-nine he contracted venereal disease, which was probably syphilis. His illness had commenced four months before admission with pain in both kidneys, especially the left; during the few days before he

Digitized by Google

entered the hospital the pain had run down into his left testicle. On admission he was found to be suffering from a good deal of pain in his left loin, and it was intensified by pressure or movement. The left testicle was swollen and tender, but was not retracted. The pain extended from the loin to the front of the abdomen and thigh. The urine contained neither blood nor albumen. There was tenderness over the left renal region in front as well as behind, but no definite tumour was felt. On the 19th he had severe pain and cramps in front of his left thigh, the back of the left thigh, the gluteal region, and the left side of the abdomen. He then became very cold and white, lost his sight, and very soon afterwards died. The diagnosis made during life was renal calculus.

Post-mortem examination.—There was an aneurysm, the size of a walnut, arising from the arch of the aorta just before its bend, which projected backwards and to the right, and caused some flattening of the trachea. The arch of the aorta and the thoracic portion were very atheromatous. There was a large aneurysm arising from the left side of the abdominal aorta, immediately below the diaphragm. Its orifice measured 1½ inches by ¾ inch. It extended backwards and to the left, and had eroded the body of the eleventh dorsal vertebra. It had also ruptured into the subperitoneal tissue, the extravasated blood pushing forwards the left kidney and extending downwards as far as Poupart's ligament.

Abdominal aneurysm mistaken for perinephritic abscess and renal sarcoma.—An engine-driver æt. 33, was admitted on October 6th, 1891, for pain in the lower part of the back, lumbar regions, groins, and hips. Seven years before admission he had suffered from syphilis. He was first laid up in October, 1890, with a gnawing pain in his back; he kept in bed for three weeks, and was then able to return to work for six months, although he was never free from pain. At the end of this time he had to lie up again. On September 28th he attended the Out-patient Department, and a note was made in his report to the effect that nothing abnormal could be detected in his abdomen. On admission he was anæmic and rather wasted. He suffered from a good deal of pain, and could not lie in one position for any length of time. A hard, solid mass, which was moveable, could be felt in the region of his right kidney. On October 17th he suffered from insomnia and frequent micturition. On October 19th there is a note in the report by the physician: "During the last five days the tumour on the right side has become much more obvious and nearer the anterior wall; it is harder and more fixed, also somewhat larger. The tumour extends below the umbilicus half an inch, it is not fixed, but does not move with respiration. The loin is fuller, the normal depression less than previously. There is no rigidity of the muscles. For some time he has been unable to fully extend the right thigh without pain. There is no great pain on vigorous percussion of the right loin. Temp. 101°, pulse, 130. The indications are an acute inflammatory swelling of the right kidney, involving the sheath of the psoas, possibly commencing perinephritic mischief. No pus has been noticed in the urine. I noticed no renal tumour at Out-patients at the end of September." On October 22nd he was anæsthetised and the tumour was explored. A soft and easily broken-down mass was felt, and thought to be a new growth. During the exploration a large gush of blood came. The wound was packed with gauze, and he was put back to bed. Two days afterwards there was a sudden gush of about 1½ pints of blood when he was being dressed, and soon afterwards he died.

Post-mortem examination.—On opening the abdomen the right kidney, the duodenum, and colon were found to be displaced forwards, downwards, and inwards by a tumour in the right loin, which proved to be an aneurysm of the abdominal aorta. It measured 10 inches in length, and extended across the spine to the psoas muscle on the opposite side; it extended upwards to the body of the tenth dorsal vertebra, and had eroded the bodies of the eleventh and twelfth dorsal, and the first, second, and third lumbar vertebræ. It communicated with the aorta by an oval opening, 3¾ inches in circumference, on its posterior surface, just at the aortic opening in the diaphragm. It contained 14 ounces of blood-clot. Below the blood extended into the right psoas, infiltrating the muscle as far down as the iliac fossa. The kidneys were both quite healthy, and the ureters were not affected, although they were pushed forwards by the sac of the aneurysm.

When cases present so many symptoms which are usually associated with renal disease, a careful

Digitized by Google

consideration of the nature of the patient's occupation, a previous history of syphilis, of sudden strain or injury followed by pain in the abdomen or back, the presence of pulsation and of a bruit should indicate the possibility of the presence of a diffuse aneurysm of the abdominal aorta.

In all doubtful cases an examination by the X rays should be made, the points to examine for being a large pulsating area connected with the aorta, a loss of the normal contour of the bodies of the vertebræ from erosion, and in a case of renal calculus, a dark, opaque body in the position of the kidney or ureter.

When the aneurysm has eroded the bodies of the vertebræ and caused paraplegia as a result of compression of the spinal cord, the diagnosis must be made from spinal caries, from a malignant growth pressing on the cord, from myelitis, or from paralysis due to obstruction of the aorta by a thrombus or embolism.

In a case of this type a careful examination must be made for the presence of a deep-seated abdominal tumour with expansile pulsation and a bruit. When the paralysis is due to the blocking of the lower end of the aorta, there would be an absence of pulsation in the femoral arteries.

If the sac of the aneurysm presses on the cardiac end of the œsophagus, dysphagia and wasting may be prominent symptoms, and lead to a diagnosis of epithelioma of the œsophagus.

The following case was of this nature:

Aneurysm of the abdominal aorta rupturing into the mediastinum, and forming a circumscribed tumour pressing on the œsophagus; rupture into the pleural cavity; diagnosed cancer of the œsophagus.—An asylum attendant æt. 30, who had served for twelve years in the Dragoon Guards, was admitted on November 8th, 1871, under the care of Dr. Owen Rees, for wasting and weakness. He had previously suffered from rheumatism, gonorrhœa and syphilis. His illness had come on in an insidious manner about six months before, with pain between the shoulders, wasting, and loss of appetite. His bowels had been constipated, and for several days he had not been able to retain any food, anything he attempted to swallow, even fluids, being rejected. He stated that the food seemed to get halfway down and was then returned at once. On admission he was emaciated. The abdomen was retracted, the muscles were rigid, but no tumour could be felt. There was no tenderness. He expectorated watery-looking sputum, which was tinged with blood. He could only take a little ice to suck, and he gradually became weaker and died on the 12th. He was thought to be a case of epitheliomatous stricture of the œsophagus.

Post-mortem examination.—Both pleural cavities contained a good deal of blood. A mass of scarcely coagulated black blood, about the size of a hen's egg, lay in a cavity between the base of the lung and the upper surface of the diaphragm, apparently formed by the accidental absence of adhesions at this spot.

Just below the diaphragm there was an aneurysm, the size of an orange, springing from the anterior wall of the aorta, and communicating with it by an oval aperture about 1¼ inches long. The sac contained a good deal of laminated clot, and from its upper edge an aperture, which would easily admit two fingers, led upwards through the diaphragm into a cavity about the size of a cricket ball, which had hollowed out the posterior mediastinum and pushed the heart forwards, and the aorta and œsophagus backwards. It was this condition which had caused the dysphagia.

The following case also illustrates the difficulty which may arise in making a correct diagnosis:

Abdominal aneurysm; first diagnosed lead colic, then aneurysm, and later cirrhosis of the liver and malignant disease.—A painter, æt 33, was admitted on April 7th, 1891, for abdominal pain. He had served his time in the army, and had suffered from both gonorrhœa and syphilis. Seven months before admission he began to suffer from a dull, aching, throbbing pain in the abdomen, which was referred to a point about an inch to the left and below the umbilicus. It was also felt in the back, was continuous, but worse at times, and was much increased by exercise. He consulted a doctor, who diagnosed *lead colic*, but the pain grew worse, and he was then told that he was suffering from aneurysm. On admission there was increased resistance, and impaired resonance in the left hypochondriac region; no tumour could be felt, but on deep pressure a distant pulsation could be detected. Over an area of 1½ inches in diameter, above and to the left of the umbilicus, a soft blowing systolic murmur was audible. He was treated with potassium iodide, gr. v, t. d. s.,

Digitized by Google

and went out relieved on June 9th, 1890. On August 3rd he was again admitted, and found to have an enlarged liver and jaundice. A diagnosis of cirrhosis of the liver was suggested. He suffered a good deal of pain, became very emaciated, and died in a semi-comatose condition on September 15th a diagnosis of malignant growth in front of the aorta with secondary growth in the liver having been made.

Post-mortem examination.—There was an aneurysm of the abdominal aorta about the size of a cocoa-nut situated behind the pancreas and stomach. The orifice measured 3 inches by 2 inches, and was filled with laminated clot, except at its lower angle, where there was a valvular opening leading into the cavity of the aneurysm. The sac was filled with partly decolourised blood-clot. The peritoneal cavity contained about two pints of thick puriform fluid and flakes of lymph. The heart weighed 17 ounces, and was much hypertrophied and dilated. The valves were healthy. The left kidney weighed 3½ ounces, the left renal artery being completely blocked by an ante-mortem thrombus. The cœliac axis arose from the front of the aneurysm, and its orifice was quite impervious. The superior mesenteric came off from it a little further down, and its canal could be traced into the part of the sac which contained recent clot. There was no sign of malignant disease.

This case is of great interest, for severe abdominal pain occurring in a painter led to a diagnosis of lead colic, but a subsequent examination enabled the doctor to diagnose aneurysm. The symptoms which developed afterwards, however, were very misleading, so that he was considered to be suffering from cirrhosis of the liver, and finally, on account of marked wasting, from malignant disease.

The diagnosis of the rupture of an abdominal aneurysm must be made from other conditions which cause sudden abdominal pain and collapse, *e. g.* perforative peritonitis, acute intestinal obstruction, acute hæmorrhagic pancreatitis, ruptured extra-uterine fœtation, etc.

PROGNOSIS.—The prognosis is extremely grave. I only found two cases of cured aneurysm of the abdominal aorta out of 18,678 necropsies, the cause of death in one being cerebral hæmorrhage, and in the other aortic incompetence. It is, therefore, essentially a disease which kills.

The average duration of life, from the time the aneurysm first becomes manifest, is about thirteen months. The shortest history of the disease in the series of fifty-four cases was a week, and the longest twelve years.

Death is frequently sudden from rupture of the sac into the peritoneal, pleural, or pericardial cavities, hollow viscera or lungs, but when the rupture occurs into the retro-peritoneal tissues death is generally much slower. Death occurred from starvation and exhaustion in the case above mentioned, in which the œsophagus was compressed. If the renal arteries become obstructed uræmia may supervene. Death may occur independently of rupture, and is then found to be due to exhaustion and cardiac failure, or it may be caused by some intercurrent disease.

TREATMENT.—The treatment of this terrible disease is unsatisfactory and discouraging. A most comprehensive paper has been written on the treatment of aneurysm by Dr. Guy L. Hunner, and it commences thus :—"The study of the literature concerning aneurysm of the aorta convinces me that this dreadful malady is usually a surgical disease."* I must confess that I have never seen a case cured by either medical or surgical treatment. The possiblility of a spontaneous cure can be practically disregarded—a statement which is borne out by the fact that only two examples were found in a series of 18,678 post-mortem examinations.

Complete rest, and a diet very much limited, both in solids and fluids (*e. g.* 8 ounces of fluid and 10 ounces of solid food). The so-called "Tufnell" treatment holds out very little hope to the patient of ultimate recovery or even of permanent benefit, so that the result which is usually obtained is hardly commensurate with the extreme tediousness and distress occasioned by the resulting hunger, thirst, and general discomfort. I shall never forget the graphic account a patient gave me of her sufferings whilst she was undergoing this treatment during the hot season in Calcutta.

The following case illustrates an attempt at dieting on the lines suggested by Tufnell :

Aneurysm of the abdominal aorta and superior mesenteric artery; thrombosis of the left renal vein.—A married woman æt. 32, was admitted on May 1st, 1874, under the care of Dr. Pavy, for a

* Aneurysm of the aorta treated by the insertion of a permanent wire and galvanism (Moore-Corradi method).

pulsating tumour in her abdomen, which she had first noticed about five months before. The pulsation was most marked when she was lying down. On admission she complained of the pulsating tumour and the pain it caused, which radiated from the bodies of the lumbar vertebræ to the front of the abdomen, and sometimes extended down her right leg. Her easiest position was sitting up with her body doubled forwards. There was a large pulsating tumour, occupying the epigastrium and upper part of the umbilical regions, which was spherical in shape, and measured about three inches in diameter. The pulsation was expansile, and a well-marked systolic bruit was audible over it. The urine was 1020, and contained a good deal of albumen. She was ordered the following diet:

Breakfast: Bread and butter, 2 ounces. Milk, 2 ounces.

Dinner: Boiled or broiled meat, 4 ounces. Claret, 4 ounces.

Tea: Bread and butter, 2 ounces. Milk, 2 ounces.

She suffered from intense pain, constipation, and insomnia, and on May 10th had a series of epileptiform fits and died.

Post-mortem examination.—There was an abdominal aneurysm which extended across the front of the spine, and for an inch or more to the right of it. The vena cava was pushed off the spine on the outer side of the aneurysm, but its canal was quite patent. The left renal vein was plugged by an ante-mortem clot which was softening. Anteriorly the aneurysm had pushed forwards the pancreas, duodenum, and pyloric end of the stomach, and the stomach was rather dilated. The opening of the aneurysm was on the anterior wall close to the superior mesenteric artery. The wall of the sac was thin, and lined with pale and soft clot.

Balfour's modification of Tufnell's treatment by the administration of potassium iodide, in doses from 10 to 30 grains, is equally unsatisfactory, and, as Hunner writes, "to be properly carried out requires first a patient of more than ordinary hardihood, and secondly, conditions with which it would be impossible to surround the average patient." Acetate of lead, aconite, ergotine, and calcium chloride are other drugs which have been recommended, but are equally disappointing. Morphia is generally essential for the relief of pain, and should not be spared in the treatment of such a terrible and almost hopeless malady. Subcutaneous injections of a sterilised 2 per cent. solution of gelatine into the gluteal region was first introduced by Lancereaux for the treatment of aneurysm, in the hope of producing ante-mortem thrombosis in the sac, but the results have not been very satisfactory. The greatest possible care must be exercised in the preparation of the gelatine solution to render it absolutely sterile, as tetanus has followed the injection of this substance in several cases.

Instances of cure brought about as a result of pressure on the abdominal aorta have been recorded, but are few and far between. It is certainly most exceptional to get such an excellent result as that recorded by Murray. The pressure may be exerted on the proximal or distal side of the aneurysm, according to its position. When the aneurysm arises from the lower half of the abdominal aorta, proximal pressure may be resorted to; but as in nearly 70 per cent. of the cases the sac comes off from the aorta in the immediate neighbourhood of the cœliac axis, distal pressure is the only method of applying this form of treatment, the object of which is to promote the formation of adherent thrombi in the sac. The bowels should be well opened and an anæsthetic administered. The pressure may be applied by means of a Lister's tourniquet, but the greatest possible care must be exercised on account of the possibility of inducing enteritis or peritonitis, and precautions must be taken to avoid any pressure on the renal arteries, as compression of these vessels may lead to suppression of urine. This method of treatment is also unsatisfactory, and is well illustrated by the following case:

Aneurysm of the abdominal aorta involving the origins of the cœliac axis and superior mesenteric artery; death from peritonitis induced by pressure with a clamp.—A dock labourer æt. 30 was admitted on February 9th, 1872, under the care of Dr. Wilks, for abdominal pain. He had served in the army, and in 1885 contracted syphilis. Six months before admission, when walking to his work in the morning, he noticed a sharp pain in the middle of his abdomen, which obliged him to sit down and rest. He had also suffered from pain in his

Digitized by Google

testicles, especially the left. On admission there was a pulsating tumour extending from the epigastric region into the left hypochondrium, which was dull on percussion, with a strong heaving impulse and lateral expansion. On ascultation a double blowing sound could be heard over it, nearly synchronous with the two sounds of the heart. The urine was normal. On February 15th he was seen by Mr. Thomas Bryant, who advised distal pressure with Lister's clamp. On February 20th chloroform was administered at 2 p.m., and a large screw clamp was placed on the abdomen, compressing the aorta immediately above and to the left of the umbilicus. The femoral pulsation was not completely controlled, and at 3 p.m. the tourniquet was reapplied, as it had slipped. At 3.30 p.m. he began to retch. At 4.30 p.m. the tumour seemed harder, but the systolic bruit was still audible. At 8.30 p.m. the pulsation in the aneurysm was less strong. At 12.15 a.m. the stomach was found to be much distended; at 2 a.m. the tourniquet was removed, but the tumour was found to still pulsate strongly. At 2 p.m. the tourniquet was reapplied, and about 5 p.m. the legs became cold and the pulse intermittent. The tourniquet was removed at 6 p.m., as the patient appeared to be so weak. The pulse was 126 at 8 p.m., and he was only semi-conscious and passed his motions under him. He gradually became weaker, and died at 5 a.m. on February 22nd. About 6 oz. of chloroform and 14 oz. of A.C.E. mixture were used when the tourniquet was first applied, and 4 oz. of chloroform on the last occasion.

Post-mortem examination.—There was an aneurysm of the abdominal aorta about the size of an orange, which arose from the aorta between the orifice of the cœliac axis and the superior mesenteric, and which included both the orifices of these vessels. The opening from the aorta into the sac was about the size of a halfpenny. The renal arteries were flattened. The sac contained an ante-mortem adherent laminated clot, which was about one third of an inch in thickness. The rest of the aorta was quite healthy. The pressure during life had been exerted on the transverse colon and mesocolon, and on the root of the mesentery. Just at the bifurcation of the aorta a coil of ileum, four feet below the duodenum and its adjacent mesentery, had been involved. The part of the ileum, above the seat of pressure was thickened, dilated, darkened, and full of fluid, whereas below the bowel was not thick and was empty. There had evidently been obstruction as a result of paralysis of the gut following the pressure. The tissues about the compressed part of the aorta were loaded with effused blood, and there was a good deal of lymph on the parts involved.

Compression usually hastens death, as it may induce peritonitis, gangrene, and obstruction of the gut, bruising of the pancreas, etc.

Ligature of the aorta has been attempted on several occasions, but has invariably proved fatal within a few hours or days.

MacEwen introduced a method of treating this disease by needling the inner lining of the sac and waiting for the formation of a thrombus; but it is unsatisfactory for aneurysms of the abdominal aorta, as it is only applicable to those cases in which the sac lies against the abdominal wall, and then there is very little hope of cure or even amelioration.

Galvano-puncture was introduced by Cinicelli, and is still in vogue in Italy.

In 1864 Mr. Charles Moore, of the Middlesex Hospital, attempted to cure an aortic aneurysm by introducing a quantity of wire into the sac. The operation was followed by marked improvement, but unfortunately sepsis followed and death occurred from this cause. The method of introducing wire into the sac has been modified by Corradi, who has added electrolysis to the procedure, and by this combined method some successful cases have been recorded by American surgeons, especially by Stewart of Philadelphia. I shall not attempt to describe the operative technique, but will mention some important points to which attention must be paid.

It is necessary to have fine wire with a good spring, so that it curls as soon as it is introduced through the needle into the sac, and which readily corrodes and forms a rough surface on the application of the continuous current. Hunner recommends a silver alloy wire containing 75 parts of copper per 1000, and of size No. 8 to No. 27 standard gauge. A continuous current battery is required, the positive pole being attached to the wire in the sac, and the negative pole to a metal plate covered with a wet towel and applied to the back. The strength of current must be from 10 to 20

Digitized by Google

milliampères, and should be allowed to run for about an hour. It is imperative to attach the positive pole to the wire in the sac, as a firm dark acid clot forms about the anode, whereas a frothy alkaline mass would collect if the kathode were used.

A needle just large enough to allow the passage of the wire should be selected, and it should be covered with some non-conducting material. Hunner recommends best French lacquer, made by Bethlen Bros. of New York, as the most satisfactory preparation. Needles thus treated can only be sterilised in dry heat, and for this purpose should be placed in a test-tube, which is corked and kept in a dry chamber at 160° C. for an hour.

Great care must be exercised in the selection of suitable cases for this method of treatment, and some of the dangers which may accrue must be considered. Evidence of general arterio-sclerosis is a contra-indication for such a method of treatment. The possibility of embolism from the breaking down of some of the laminated clot in the sac of the aneurysm, or the sudden closure of some important artery with the newly formed clot after the introduction of the wire, *e. g.* of the superior mesenteric artery, which would lead to infarction of the intestine followed by gangrene of the gut and death in a day or two, must be considered.

There is also a danger in the case of a multilobular aneurysm of the development and rupture of a secondary sac. The possibility of the wire penetrating the opposite side of the sac, or of entering the aorta, must be eliminated by the selection of a wire which will curl as soon as it is introduced into the sac. If the aneurysm is of the fusiform variety the treatment is not only useless, but will shorten the patient's life.

Hunner in his paper gives the result of an analysis of twenty-three cases of aneurysm of the aorta, seventeen thoracic and six abdominal, treated by the Moore-Corradi method. Of these, four, or 17 per cent. (three thoracic and one abdominal), were cured. One patient (Rosenstirn's) was alive eleven years after the operation; another (Stewart's) was shown, by an autopsy three years after the operation, to have been cured as far as the aneurysm was concerned; Kerr's patient was seen working as a street pavior ten months after, and Noble's patient lived for eight months with no sign of his abdominal aneurysm, and died from an entirely different cause. In nine, or 39 per cent., of the cases life was prolonged and the symptoms were relieved, whereas in the remaining eight death was probably hastened.

An unbiassed consideration of the various methods recommended for this fatal disease shows that the combined method, the introduction of wire and electrolysis, holds out the greatest hope of cure, provided that the form and the position of the aneurysm are suitable. For further details of the treatment, and for an account of the cases treated in this manner, I must refer you to Dr. Hunner's excellent paper.

Spurious Gastric Diseases.—The difficulty of always recognising the nature of the apparently gastric symptoms is noticed by Jones, who remarks on the various causes of pain in the epigastrium, and the need in all cases of gastralgia of a differential diagnosis between the rather rare essential gastralgia and cholelithiasis. In the absence of symptoms and physical signs of gall-stone colic, the diagnosis is not always readily made. Vomiting is not a sure symptom of gastric disease. It may be due to cholecystitis, uræmia, or other causes. Appendicitis in its various phases and effects needs attention. Reflex hyperchlorhydria may confuse the diagnosis. Pancreatic disease is another condition which, while relatively uncommon, may be confusing. Obscure tubercular peritonitis that causes no tumour may give rise to gastric symptoms. Heart lesions may also simulate gastralgia. Arterio-sclerosis, arterio-spasm, and syphilis may also cause confusion, but the use of the iodides in the latter case will show the nature of the condition. There may also be true sclerosis of the arterioles of the stomach, causing ulceration, or diaphragmatic pleurisy may incite reflex pain in the gastric region, and only close study will determine the true nature. In his experience, he says, pain in the stomach is very rarely the result of pelvic disease, while vomiting is a common result. Disease of the pelvic organs is one of the causes of spurious gastric symptoms. Lastly, he mentions eye-strain as a cause of various stomach symptoms. —*Journ. A. M. A.*, October 31st, 1903.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 579. WEDNESDAY, DECEMBER 2, 1903. Vol. XXIII. No. 7.

CONTENTS.

PAGE

*A Lecture on Some Cases of Gastric Surgery and their Results. By D'ARCY POWER, F.R.C.S. 97

*On Some Aural Cases. By STEPHEN PAGET, F.R.C.S. 107

*Is an Eczema Ever Symptomatic? By T. ROBINSON, M.D. 111

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

A LECTURE

ON

SOME CASES OF GASTRIC SURGERY AND THEIR RESULTS.*

By D'ARCY POWER, F.R.C.S.Eng.,

Senior Surgeon to the Victoria Hospital for Children, Chelsea, and Senior Assistant Surgeon at St. Bartholomew's Hospital.

GENTLEMEN,—The object of these occasional lectures is to enable us to bring before you from time to time the subjects which are engaging our special attention in medicine and surgery. To all of us who are much occupied in hospital work there come, as if by accident, series of cases which lead us to attain special skill in diagnosis or treatment. Since I have been attached as assistant surgeon to St. Bartholomew's Hospital I seem to have had an unusual share of cases requiring operations upon the stomach. When I was asked, therefore, to lecture to you this evening, I thought it might be to your advantage, as well as my own, if I put together the scattered records of these cases and drew from them such conclusions as seem to be justified by the results.

The importance of the surgery of the stomach is likely to increase in the future, because the organ lends itself readily to manipulation, and because it is subject to several pathological conditions which can be greatly benefited by operation. I make no apology, therefore, in lecturing to you this evening upon gastric surgery, or for bringing before you the results which I have obtained. But as I do not propose to weary you with the details of cases, I have made an abstract (see page 105), and when it is necessary to refer to them I shall do so by numbers. (Further details of these cases are published in the 'St. Bartholomew's Hospital Reports,' vol. xxxix, 1903, p. 19.)

* Delivered at the Medical Graduates' College and Polyclinic, November 2nd, 1903.

Digitized by Google

The operations which I have performed upon the stomach are divisible into two great groups: those done on the spur of the moment for acute perforation, and those done more deliberately—like gastrostomy and gastro-enterostomy—for the relief of obstruction situated either at the pylorus or in the duodenum. I do not intend to take gastrostomy into consideration this evening, nor have I included it in my list of cases, though as surgeon in charge of the throat department at St. Bartholomew's Hospital I have to perform the operation frequently on patients who come with the symptoms of cancer of the œsophagus.

I. Acute Perforation.

Turning first to the cases of acute perforation, you will see that I have been obliged to include acute perforation of the duodenum with acute perforation of the stomach, as the symptoms are very similar, and there is no means of distinguishing between the two until the abdomen is opened. The table shows you that I am basing my observations upon seven cases of acute gastric perforation, four in women, three in men; and four cases of duodenal perforation, all occurring in men.

The diagnosis in the acute cases may be beset with considerable difficulty if too much reliance be placed upon the classical symptoms of pain, vomiting, rigid abdomen, and collapse. Indeed, the house physician under whose care the case usually comes first, thinking of colic and many conditions about which I know nothing, always asks anxiously, "Why are you going to operate? Are you sure there is a perforation?" And I always give the same reply, "I am going to operate because the patient has had an attack of very acute and sudden pain, because the pulse is increasing in rapidity from hour to hour, because there is local abdominal tenderness, and because the patient looks very ill." I do not care whether or not there have been preceding symptoms of indigestion, and the vomiting may or may not be an important symptom.

The great secret of the diagnosis lies in seeing the patient early. He has not then forgotten the severity of the pain; his stomach is still tender if it be pressed upon, and there is local abdominal rigidity. After a few hours all these conditions are masked by the general peritonitis. Fortunately for the patients, the pain is so severe at the moment of perforation that they recognise it as more than an attack of indigestion, and they seek relief as soon as possible, though they sometimes wait a few hours in the hope it will improve. Self-applied remedies, too, make the pain worse. Thus one of my patients (No. 2) was attacked with pain on his way home from work, and sought to relieve it by drinking a pint of milk and soda-water, which led to such an increase that he asked the first policeman to take him to the nearest hospital; whilst another (No. 10) took a glass of strong brandy and water with a similar result. Still, people feel pain very differently, and too much reliance must not be placed upon this symptom, especially if it is felt to be less when the patient has been put to bed comfortably.

The condition of the abdomen is also a matter upon which we should be careful. We are accustomed to think of it as rigid, motionless, and either retracted or distended. But experience teaches that these symptoms are only marked in the later stages, and it is very important that the condition should be recognised before they become manifest. Cases 3, 5, 8, 9, 10, and 11 show that the abdomen may remain soft and may move freely in spite of an acute perforation, but this absence of rigidity seems to be more common in acute duodenal than in acute gastric perforation. Soon, however, peritonitis sets in, the symptoms merge into those connected with the inflammation of the whole peritoneum, and in the absence of an exploratory operation the patient dies without the cause being discovered.

But even when the patient has applied for relief quite early, I prefer to wait for a short time before the abdomen is opened, and in this interval I have the pulse counted carefully by the same person every half-hour. If the pulse-rate rises I operate without any hesitation; if it sinks or remains stationary I hold my hand, and in this way I believe that we have avoided several unnecessary operations.

But if the diagnosis of acute perforation of the stomach is not always quite easy, the recognition of a perforated duodenal ulcer is still more difficult. This perforation is situated deep in the abdominal cavity, and the patient appears to have a difficulty in locating the exact situation of his pain until the extravasated material has trickled into his right iliac fossa and has set up some irritation of the

Digitized by Google

parietal layer of the peritoneum. He then complains of pain in the right iliac fossa, which is easily mistaken for an attack of acute appendicitis. I made this mistake in one of my cases (Case 11), and explored the vermiform appendix before I recognised the nature of the lesion. This patient was the only one of the four who recovered, and I attributed his good fortune in part to the free exposure and subsequent good drainage. One of the other cases (Case 8) had such obscure symptoms at the time of his admission to the hospital that he was thought to be suffering from pneumonia, and the error was only discovered when the onset of peritonitis cleared up the diagnosis. The abdomen moves more freely during respiration after the perforation of a duodenal ulcer than after rupture of a gastric ulcer; and it is perhaps noteworthy that all four of my cases occurred in men.

The treatment of acute perforating ulcer is clear and simple. The abdomen must be opened as soon as the diagnosis is tolerably certain, and the perforation must be closed. I say when the diagnosis is tolerably certain, because I think it is better to err and make an unnecessary exploration rather than to wait with excess of caution and allow a perforation to continue and discharge the contents of the stomach or duodenum into the peritoneal cavity. The diagnosis is complete as soon as the peritoneum is opened, for gas escapes at once if there is a perforation. In such a case the stomach is drawn out of the wound, which I make for greater convenience through the left rectus abdominis rather than in the middle line. In men I examine first the pyloric end of the stomach, and in women the cardiac extremity first and always the anterior wall. The hole is easy to recognise, and it is usually surrounded by indurated tissue. In my early cases I used to be too careful, and wasted time in suturing this tissue, for the stitches very readily cut out; now I shorten the operation by boldly bringing together the stomach walls where they are healthy, and I do not much mind how large a piece of the stomach is invaginated. The value of this method was amply shown in Case 7, for the woman had a hole in her stomach as large as a sixpence, whilst the surrounding gastric wall was thickened and infiltrated for a good three inches round the perforation. If I had treated her at all in a "niggling" fashion she would certainly have died from continued leakage; as it was I turned in all the thickened area, and she left the hospital cured within five weeks of the operation.

The treatment of an acutely perforated duodenal ulcer is similar to that for a ruptured gastric ulcer, but the technique is certainly more difficult. The perforation is generally situated in the first part of the duodenum near the pylorus. It is distinguishable from a gastric ulcer by the small, well-circumscribed hole, and by the constant flow of thin fluid, either clear or bile-stained, which issues from it. The fluid wells up with some force, and is quite different from that which comes from a gastric ulcer. But the hole is awkwardly placed deep down in the abdominal cavity, and often with the liver hindering the free use of the needle. It is much more difficult, therefore, to close it securely than to sew up a perforated gastric ulcer, though they are sometimes uncommonly awkward when the ulcer is situated on the posterior wall of the stomach.

The prognosis is fairly good in the case of acute gastric perforation; bad in cases of perforated duodenal ulcer. In six out of seven cases of gastric perforation the hole was entirely closed; five of the seven patients left the hospital cured, one died of septic pneumonia nineteen days after the operation, and one died soon after the operation because I had not closed the opening securely. It was far otherwise with the cases of perforated duodenal ulcer, for I was only able to save one out of four cases, and only in two cases did I succeed in closing the perforation completely.

The after-history of the patients in the four cases in which I have succeeded in tracing them was:—Case 1.—The patient was alive, well, and remained free from dyspepsia two years and seven months after the perforation. Case 2.—The patient remained free from pain for twelve months after the operation, except on one occasion, when he confessed that he had supped somewhat too freely on "whelks and trotters." After twelve months he again began to suffer from dyspepsia, but as he was a printer's labourer, aged thirty-four, his habits were not very regular. Case 4.—The patient was well and free from dyspepsia six months after the operation. Case 11.—The cured duodenal ulcer was a typically healthy man a year after the operation, and had suffered from no dyspeptic troubles.

Digitized by Google

II. Gastro-enterostomy.

I now come to the second part of my subject, or those operations upon the stomach which are done deliberately. These operations are more interesting and require somewhat more skill. The finding and sewing up of a perforation is urgent, but not very difficult. The performance of a satisfactory gastro-enterostomy is by no means so easy a matter, especially if it be done, as I think it ought to be done, by direct suture rather than by means of a button; whilst we are not yet all agreed as to the cases in which it will yield the best results.

The general plan of the operation of gastro-enterostomy is to secure a communication between the stomach and the jejunum, leakage being prevented by suturing the stomach and intestine together not only at the actual point of anastomosis, but also at such a distance as will allow of some extent of the two peritoneal surfaces being brought into contact. There are at least three methods of uniting the stomach to the upper part of the jejunum: McGraw's, the pre-colic, and the post-colic operations. The early stages are identical in each operation. The abdomen is opened on the left side by a vertical incision about three inches in length, situated above the level of the umbilicus and in the middle of the rectus abdominis muscle. The fibres of this muscle are separated and retracted until the posterior sheath is fully exposed in the whole length of the wound. The posterior layer of the rectus sheath and the fascia transversalis are then divided, oozing is stopped, and the peritoneal cavity is opened, also in the whole extent of the wound. It saves time at a later stage of the operation if two pair of pressure forceps are put upon the cut edges of the peritoneum. The stomach is first drawn out and is examined systematically, but too evident symptoms of disease are not to be looked for in every case. There may be extensive disease of the mucous and submucous coats, and yet the serous and muscular layers may remain unaffected. A loop of small intestine is then picked up from the left side of the abdominal cavity, and is rapidly traced upwards until a fixed point is found. This is the duodeno-jejunal angle, and it marks the end of the duodenum and the beginning of the jejunum. The surgeon knows that he is tracing the small intestine in the right direction if the calibre becomes smaller as he proceeds, for the lower parts of the jejunum and ileum have a greater diameter than the duodenum and the upper part of the jejunum. Care must be taken not to over-stretch the jejeunum by joining the stomach too near the fixed duodeno-jejunal angle, or the tension may produce disastrous vomiting when the operation is ended. In fact, I believe that this is one of the main causes of the "vicious circle," which is so greatly dreaded by the French surgeons that they have suggested a second anastomosis between the jejunum and a more distal loop of intestine. To prevent this tension I always try to suture the stomach to the jejunum nine inches away from the point where the intestine can no longer be pulled upwards, whilst the suturing is done from left to right to prevent any encroachment on the distance thus left. From this point the three operations diverge.

McGraw's operation of gastro-enterostomy consists in allowing an elastic ligature tied tightly to cut its way through the coats of the stomach and intestine at points which have been isolated by peritoneal adhesions from the general abdominal cavity. I adopted the method in Case 12, as the patient was old and very feeble. It was eminently successful, but the method did not seem to be superior to the ordinary direct suture. The stomach and a loop of jejunum are united by a continuous suture of fine silk for a distance of three inches, the stitches passing through the serous, muscular, and part of the submucous coats in each case. A straight Hagedorn needle carrying a specially made piece of round rubber is then driven through all the coats into the cavity of the stomach by a stab made at right angles. The needle is carried along within the cavity of the stomach for an inch, and is then brought out vertically through the walls, carrying with it the rubber ligature. The needle is then made to transfix the small intestine at a point corresponding to that at which it emerges from the stomach. It passes along the lumen of the jejunum for an inch, and is again driven through the intestinal wall at a point corresponding to that where it first entered the stomach. The elastic ligature now lies both in the stomach and in the jejunum, one end lying outside the stomach, the other end outside the small intestine. The two ends of the ligature are then tied in a reef-knot as tightly as can be done without undue puckering of the tissues, and to prevent the knot slipping the ends of the ligature are

Digitized by Google

bound together with two ligatures of silk. The ends of the elastic ligature are then cut short, and the area of operation is shut off by uniting the stomach to the jejunum by a continuous suture placed half an inch in front of the elastic ligature. The pressure exercised by the elastic ligature on the coats of stomach and intestine establishes a fistula in the course of a few days, but not before sufficient peritoneal adhesions have been formed to prevent leakage.

The pre-colic operation of gastro-enterostomy consists in finding the stomach, bringing up to it the first loop of the jejunum, and uniting the two by a continuous suture in front of the transverse colon. An opening is then made in the stomach and in the adjacent loop of jejunum, the mucous membrane is cut away where it protrudes with a pair of curved scissors, and the edges of the two wounds are sewn together with a continuous suture of silk which passes through all the coats of the stomach and the jejunum, beginning posteriorly and, as before, on the left side. This suture is called the hæmostatic suture, because it stops the oozing of blood from the cut edges of the stomach and intestine. But it does not always do so completely, and any bleeding point should be picked up and tied. A row of Lembert's sutures, placed anteriorly, shuts off the fistula, and, by bringing together the gastric and intestinal surfaces of peritoneum, soon protects the wound, for we know that aseptic peritoneal adhesions are practically watertight in eighteen hours after their formation.

The post-colic operation of gastro-enterostomy proceeds at first along the same lines as the two operations just described; but when the stomach and first part of the jejunum have been found they are gently put aside, and the transverse colon is drawn out of the wound and turned upwards to expose the mesocolon. A hole is made in the transverse mesocolon large enough to expose a sufficient portion of the posterior wall of the stomach. The subsequent steps of the operation are the same as in the pre-colic method, except that I usually prefer to cut away the mucous membrane which protrudes from the incisions in the stomach and jejunum rather more freely than in the previous operation. The operation thus takes a little longer than either McGraw's or the pre-colic, but the result is more satisfactory, for the fistula lies deeply in the abdominal cavity, covered by the transverse colon, and it is thus less subject to tension.

Each operation fulfils its purpose of making a satisfactory short circuit, but McGraw's operation and the pre-colic method seem to be attended by somewhat less shock than the post-colic operation. The patient upon whom I performed McGraw's operation (Case 12) certainly suffered more pain than any of the others. This operation, too, has the disadvantage of introducing a foreign body into the abdominal cavity, and it does not take a materially shorter time than an ordinary gastro-enterostomy, so that it does not possess any great advantage for one who is at all skilled in abdominal work. At least two sets of sutures are required, and, although the puncture holes in the stomach and intestine are filled by the elastic ligature surrounded by the swollen mucous membrane, there is a possibility of leakage, so that the operation is not to be recommended to those who are less sure of being able to make a satisfactory anastomosis by direct suture. The pre-colic operation is attended by less shock because there is less disturbance of the abdominal contents than in the post-colic operation, and if a sufficient length of jejunum be taken there should be no fear that the colon will become constricted where it passes behind the seat of operation. There is, however, a danger that the colon may become distended as a result of intestinal paresis, and may by its pressure cause the sutures in the anastomosis to cut out. I believe that this accident happened in Case 18, though unfortunately it was impossible to get a post-mortem examination. But in this case the stomach was so matted to the surrounding tissues by adhesions that a post-colic operation was out of the question. Still the pre-colic operation might sometimes be serviceable in cases of collapse and weakness where it is desirable to finish as speedily as possible.

The post-colic operation is in every respect the most satisfactory when it is well done. The stomach and jejunum afterwards bear a fairly normal relation to each other; the colon can neither compress nor be compressed, and the anastomosis lies deeply in the abdominal cavity. As I have already said, this method has the great objection of causing a prolonged exposure, as the operation cannot be done much under forty minutes; and not only to exposure, but also to

Digitized by Google

some tension, both upon the stomach and the jejunum, so that collapse and vomiting are more frequent after this than after the other forms of gastro-enterostomy.

I have been interested to ascertain whether the size of the hole makes any appreciable difference to the patient's comfort and the success of the operation. In each case, therefore, I have varied the length of my incision into the gastric and intestinal walls. In Case 12 the distance between the points of insertion and exit of the elastic ligature was a full two inches; in Case 13 the incision was only half an inch in length; in Case 14 it was an inch and a half; in Case 15 it was three inches; in Case 16 it was an inch; in Case 17 it was three quarters of an inch; and in Case 18 it was an inch and a half. The patient (Case 13) who had only a half-inch incision reports himself to me some months after the operation (see CLINICAL JOURNAL, October 21st, 1903, p. 13) as a healthy man, free from pain and vomiting, able and willing to do a day's work. On the other hand, the patient who had a three-inch incision (Case 15) felt so hungry seven days after the operation that she stole a bun from her neighbour in the next bed and ate it—to her own satisfaction, but the nurse's dismay. It did her no harm, and it taught us that in some cases at any rate we were over-careful in dieting patients after the operation of gastric enterostomy. I show the specimen from Case 17, as the patient unfortunately died of exhaustion six days after the operation. You will see that the union is in every way most satisfactory, the point of anastomosis being exactly where it should be—as near the pylorus as possible,—for you will see from the table that the operation was performed for constriction of the duodenum due to adhesions the result of long-standing inflammations in the region of the gall-bladder. The incision at the time of the operation I estimated to be three quarters of an inch in length. You will see that it has become a round hole sufficiently large to admit a thick glass rod. So that with this aperture as an auxiliary to the constricted duodenum the contents of the stomach should have passed readily into the small intestine, and the patient would have been well nourished. I do not think, therefore, that the size of the anastomosis is of much importance, so long, of course, as it is reasonable and not a mere pin-hole.

The table (Nos. 12 to 18) shows the cases of gastro-enterostomy which I have performed. They resolve themselves broadly into cases of painful dyspepsia, with or without dilated stomach, when other means have failed to cure; and cases of emaciation, which are themselves divisible into two groups, the emaciation in one set of cases being due to mechanical obstruction to the passage of food through the duodenum, and in the other group to malignant disease of the stomach.

Here is the history of one of the cases of painful dyspepsia without dilated stomach. It serves as a type. A domestic servant, æt. 30, had suffered from attacks of severe dyspepsia for three years. The pain came on immediately after her food, and was relieved by vomiting. Sometimes the vomiting occurred directly after taking food, but often it did not begin until two or three hours afterwards. Fluid as well as solid food made her sick. She was habitually constipated, and had been anæmic ever since she was seventeen years old. She had been an in-patient at a large general hospital from January to March, and got better whilst she was dieted, but as soon as she went out to service again all her previous symptoms reappeared. An examination of her abdomen merely showed an area of marked tenderness over the site of the stomach, and the tenderness extended downwards along the region of the descending colon. I operated upon her; she recovered, and has been free from dyspeptic symptoms ever since. The stomach appeared to be perfectly healthy from the outside, but I have little doubt that she had a chronic though superficial erosion of the gastric mucous membrane. A surgeon is justified in operating upon such cases, because the recurrent attacks prevent the patient from obtaining a livelihood, and it is right, therefore, that she should undertake the risk in the hope of a cure.

The cases of painful dyspepsia with dilated stomach are very interesting and are most satisfactory. They occur in older people, and I feel sure they are often mistaken for cancer of the stomach. It is certain that I fell into this error in Case 12; I escaped it in Case 13, because the history gave a clue to the condition; whilst in Case 16, with the experience of the two previous cases comparatively fresh in my memory, I arrived at a correct diagnosis, which was unfortunately verified at the post-mortem examination.

Digitized by Google

Here is a brief history of Case 12. A man æt. 69 said that he had suffered severely from dyspepsia for the last five years, and that for two years past he had pain after his food and vomiting. During the six or seven months preceding his admission to the hospital there had been streaks of blood occasionally in the material vomited. The attacks of vomiting usually took place once in two days, and he would bring up as much as two pints at a time. He had lost flesh rapidly for the last two months. It was noted on admission that he was a thin and wasted man, with a flaccid abdomen, which moved freely during respiration. The stomach was greatly dilated, as the lesser curvature lay about three inches below the costal margin, and the organ occupied part of the epigastric, all the umbilical, a great part of the hypogastric, and some of the iliac regions of the abdomen. A succussion splash was very distinct, and a hard rounded tumour about the size of a Tangerine orange could be felt in the middle line, sometimes above and sometimes below the umbilicus. A test meal showed the presence of free hydrochloric, lactic, and butyric acids, with albumoses. I made very sure that he had cancer of the pyloric end of his stomach, and told him that, although I could not cure him, I could make his life less miserable by an operation. He consented, and when I opened the peritoneal cavity I found that the pylorus was thickened, but that the swelling which had been felt through the abdominal wall was a mass of adhesions surrounding the duodenum and attaching the whole of its first part to the neighbouring tissues. I accordingly altered my diagnosis from cancer of the pylorus to cancer of the pancreas, or perhaps of the gall-bladder. The patient made a good recovery and soon became himself again.

He writes to me under the date October 28th, 1903, after compliments, as follows:—"I hardly know how to begin, so presume I had better start from our first interview, which was on July 23rd, 1902. Went into Bart.'s August 1st. Operation performed on me August 19th. Left September 19th at my own request; went direct to a home at Reading; left there November 1st for Ilfracombe direct; bore the journey well. Out in bath-chair November 5th for an hour, and so every day when fine until December 11th, when the chair was dismissed and I walked. I can eat and digest any food. I occasionally take a glass of mild ale—no spirits, no wine. I retire to bed about ten, previously eating an apple. I rise at six, make myself a cup of cocoa—half milk,—and after washing, smoke a pipe and read until 8.30; then breakfast, my best meal; lunch at one, generally cold meat and soup. Tea at five. Dinner or supper at 7.30; seldom get meat then—usually bread and butter, cheese, salad, and plenty of onion; if fine, generally a short walk afterwards. Can walk five or six miles without feeling tired. Am out of doors as much as possible. I append my weight from time to time. July 3rd, 1902, 9 st. 4 lbs.; November 10th, 9 st. 8 lbs.; November 24th, 10 st. 8 lbs; December 8th, 11 st. 6 lbs.; December 22nd, 11 st. 7 lbs; January 3rd, 1903, 11 st. 12 lbs; January 19th, 11 st. 9 lbs.; February 2nd, 12 st. 2 lbs.; February 28th, 12 st. 6 lbs.; March 9th, 12 st. 2 lbs; April 2nd, 12 st. ½ lb.; October 28th, 11 st. 12 lbs. I have not weighed so much for thirteen or fourteen years, but remembering that I am seventy-one years old, I can't expect to keep up this weight, although I don't feel my age."

I have very little doubt, therefore, that the adhesions were due to old inflammation about the gall-bladder, probably due to the irritation of gall-stones or to the existence of a healed duodenal ulcer. I suppose that the duration of the symptoms without secondary deposits should have warned us of the mistake, but the age of the patient, the blood he brought up, and the tumour to be felt completely misled me. I have every reason to suppose that such a mistake is not uncommon, and that many people are allowed to go on vomiting and in great discomfort on the supposition that they are suffering from malignant disease of the stomach, who are in reality suffering from the effects of adhesions in the neighbourhood of the pylorus and duodenum. These patients would receive benefit from a gastro-enterostomy. The condition, at any rate, is not rare, for the post-mortem examination showed similar adhesions in Case 16; and we should certainly have said that Case 13 had a malignant stricture unless he had chosen to tell us that he dated his symptoms entirely from the time when he swallowed an ounce and a half of nitric acid. This patient must have taken a good gulp, for he had no dysphagia; his œsophagus escaped material injury, therefore, and the corrosive action of the acid seems to have been

Digitized by Google

exerted entirely upon the pyloric end of the stomach. Here, again, the anastomosis between the stomach and duodenum did the patient a world of good, and has enabled him again to perform the duties of his life in comfort. It is well, therefore, to bear these cases in mind before making too absolute a diagnosis of cancer of the stomach, and to ascertain as far as possible the condition of the gall-bladder, and whether or not there have previously been symptoms pointing to gall-stones or duodenal ulceration.

Cases 14 and 15 were domestic servants, unable to keep their places on account of recurrent attacks of painful dyspepsia; whilst Case 18 was a woman with cancer of the pyloric end of the stomach, who was dying of starvation, and she underwent an operation in the hope of gaining some benefit. Considerable difficulties attended the last operation, and Miss Carter, the nursing sister who had charge of the case, writes to me, "Operation, September 29th, at 5 p.m. Injection of coffee three ounces, and brandy one ounce, was given as soon as the operation was over. Nutrient enemata of Benger's food, Bovril, peptonised milk and brandy (eight ounces at a time) were given every six hours. The pulse improved and was very good all the next day, and the patient was comfortable until 6 p.m. on September 30th. She then got very restless, complained of feeling faint, and then fainted. She had a hypodermic injection of strychnia, and brandy was given *per rectum* with two pints of salt solution. Her pulse did not improve, and her respiration soon became sighing. The patient became restless and got very pale; her extremities were cold, but there was no sharp pain and no abdominal distension. She died at 12.45 a.m. on October 1st." In this case the stitches may have given way or, as I think is the more probable, the patient simply died of exhaustion, her death being no doubt accelerated by the stress of the operation.

The prognosis of the operation of gastro-enterostomy varies according to the class of case for which the operation is undertaken. The table shows that two of the seven cases died—one a week after the operation, with a good anastomosis; the other within twenty-four hours, probably from shock. Both these cases were women aged respectively fifty-four and fifty-two (Cases 17 and 18), worn out by previous illness, in whom the operation was undertaken as a forlorn hope. Age, however, does not preclude an operation, for Case 12, operated upon at sixty-nine, is now enjoying the reward of his pluck in a hearty old age at seventy-one. But the most satisfactory cases seem to be those where a gastro-enterostomy is performed upon young women with chronic and painful dyspepsia, for they recover easily and obtain the greatest relief from the operation.

Much of the after-treatment depends upon the feeding. We are accustomed to give nothing by the mouth for twenty-four hours after the operation, relying upon nutrient enemata every four hours. Two drachms of peptonised milk are given hourly, and if no vomiting occurs the amount is increased to half an ounce, and afterwards an ounce at intervals of two hours. Whey can often be substituted with advantage for the milk if it is found to be too heavy. The nutrient enemata consist of milk, 4 oz.; brandy, 2 oz.; Liq. Pancreaticus, 1 dr.; and sodium bicarbonate 10 gr. They are continued for four to five days after the operation, the rectum being washed out every twenty-four hours. I have thought that in some of my cases I have been somewhat too careful in dieting the patients, and in keeping them for too long a time on "slops." I am becoming somewhat more liberal, therefore, and my patients are allowed a more generous diet at an earlier period than I used to think advisable. I have found that in every case the patient himself gave the best indication of when he was over-fed, for he always vomited, and the vomiting has stopped when a smaller quantity of food was given to him at longer intervals.

These, gentlemen, are some of the cases of gastric surgery and their results which I wish to bring under your notice this evening. They show that whilst much can be done, we still have much to learn both in diagnosis and in treatment. In the cases of perforating ulcer we learn to be prompt in action, even when the diagnosis is not absolutely assured, and to trust more to the pulse and the subjective symptoms than to the objective signs. In the operation of gastro-enterostomy, on the other hand, we act with more deliberation, and pick our cases with a care which is impossible either in gastrostomy or the operation for perforated ulcer. But in every case we can only echo the pious expression of our great forerunner in surgery, "I dressed him; God healed him." It is no more possible for us than it was for Ambroise Paré to estimate the vitality or power of resistance inherent in each individual. Abdominal operations, perhaps more than all others, make the most severe strain upon the resisting force. In abdominal surgery, therefore, more than in other branches, it is especially difficult to forecast the issue of an operation. A man who is apparently strong and of fine physique will succumb where a delicate woman or a child will pull through. It is not wise, then, to promise too much, or to expect too little. Prompt action, speed, and a good anæsthetist will sometimes save even a desperate case. Gentlemen, I have done, and I desire to thank you heartily for the attention with which you have listened to me this evening.

Digitized by Google

No.	Date of admission.	Sex.	Age.	Symptoms.	Condition at time of operation.	Result.	Remarks.
				A. Cases of Perforated Gastric Ulcer Treated by Operation.			
1	Nov. 27th, 1900	F.	20	Pain after food for some months, but never sick; sudden onset of pain at 6.30 p.m.; operation at 12.30 a.m.	Pulse 105; epigastric tenderness; abdomen motionless, but not hard or distended; perforation on anterior wall near pyloric end of stomach	Cured, left hospital January 16th, 1901	Alive and well, free from dyspepsia June, 1903.
2	Nov. 29th, 1900	M.	34	Pain after food for last two years; sudden onset of pain at 8 p.m.; operation at 1.30 a.m.	Pulse 120; abdomen hard and retracted; perforation on anterior wall about an inch from pylorus	Cured, left hospital January 11th, 1901	Remained free from pain and in good condition when last seen in January, 1902.
3	Oct. 23rd, 1901	F.	17	Previous symptoms of indigestion, but had never brought up any blood; sudden onset of pain shortly after breakfast at 8 a.m.; operation at 2.45 p.m.	Pulse 132, very feeble; abdomen soft, moving freely with respiration, very tender, and more so above than below umbilicus; perforation on anterior wall of stomach 1¼ inch from pylorus; perforation large enough to admit a cedar pencil	Death from septic pneumonia on November 11th; no unfavourable symptom till November 7th	The perforation in the stomach quite healed.
4	Dec. 30th, 1902	F.	24	Pain after food for a year with frequent vomiting, but never blood; sudden onset of acute abdominal pain at 12.30 p.m.; operation 5.30 p.m.	Collapsed; pulse 108; perforation ⅛ inch in diameter on anterior wall of stomach near lesser curvature; drainage-tube for forty-eight hours	Cured, left hospital February 4th, 1903	Pleural effusion on January 12th; twelve ounces of clear fluid drawn off on January 15th.
5	April 17th, 1903	M.	37	No previous history of illness; sudden and acute pain at 6 p.m. whilst carrying timber; applied for relief 11.30 p.m.	Collapsed; pulse 156; stomach tender, but moving freely and not distended or tense; a large perforation near pylorus on posterior aspect of stomach; two drainage-tubes in median incision, and a drainage-tube passed through the abdominal wall just above each Poupart's ligament	Death seventy-six hours later	I had not completely closed the perforation; the patient died of general peritonitis.
6	July 13th, 1903	M.	23	Had felt pain for three years on left side beneath lower ribs; began to vomit six months ago, but had neither pain nor vomiting for three weeks before perforation; suddenly seized with a sharp pain at 1.30 whilst at dinner, but did not apply for relief until 6.45	Collapsed; pulse 92; abdomen tender and tense; perforation large enough to admit a full-sized probe situated near pylorus, and on anterior surface; drainage-tube three days, then a light packing of gauze	Cured, left hospital August 8th, 1903	Operation only lasted ten minutes.
7	August 17th, 1903	F.	41	Pain and flatulence for two months; vomiting after food six weeks; sudden and severe abdominal pain on the 16th, but did not apply for relief till 17th	Pulse 84; abdomen tense and tender; perforation of anterior wall of stomach near the cardiac end; the hole as large as a sixpence, and the stomach wall thickened and infiltrated for at least 3 inches round it; drainage-tube for twenty-four hours, then a plugging of gauze	Cured, left hospital September 23rd	The largest ulcer and perforation I have seen; the stomach wall was turned in very freely; there is but little doubt the patient drank.
				B. Cases of Perforated Duodenal Ulcers Treated by Operation.			
8	May 23rd, 1901	M.	41	No history of previous illness; sudden pain in epigastrium at 11 a.m.; applied for relief at 2 p.m.	Pulse 100; pain felt in right side; abdomen slightly rigid, but symptoms so ill-defined that he was thought to be suffering from pneumonia; at 12.30 a.m. pulse 120, and abdomen distended; I saw him for the first time at 2 a.m. and operated; a punched-out ulcer was found just beyond the pylorus, from which a clear fluid kept welling up	Death five and a half hours after operation	Imperfect closure of perforation; a temperate man.
9	July 13th, 1901	M.	47	No history of previous illness; sudden and very severe abdominal pain at 9 a.m., but did not apply for relief until 8.30 p.m.	Pulse 104; abdomen moving with general tenderness more marked in epigastric region; operation 10.45 p.m.; perforation at back of pylorus circular and well defined, clear fluid welling up from it; no drainage-tube	Death eight days later	Sub-diaphragmatic abscess; incomplete closure (?) of perforation; ulceration had continued; patient a drunkard; he felt so well till twenty-four hours before death that he was always asking when he might get up.

Digitized by Google

No.	Date of admission.	Sex.	Age.	Symptoms.	Condition at time of operation.	Result.	Remarks.
10	May 18th, 1902	M.	37	Stomach-ache half an hour after food for the last six months; sudden pain on right side beneath ribs and round umbilicus at 12 midday, so severe that he took a cab at once to the hospital; operation at 6 p.m.	Pulse 120; abdomen moving freely and equally; no perforation in stomach, but a small clean-cut ulcer in duodenum from which bile-stained fluid kept pouring out; aperture closed and a counter-incision made in right lumbar region, through which a drainage-tube was passed	Death 11 p.m., May 19th	The perforation was effectually closed, as it was water-tight.
11	July 26th, 1902	M.	26	Had suffered other attacks of pain; sudden onset at 7 p.m. on previous day; operation at 11 a.m.	Pulse 112; pain felt over right iliac region which was tender and full; the rest of the abdomen was moving during respiration; the appendix was found to be normal; the stomach was intact, but a perforation was discovered in the duodenum and on its anterior surface	Cured, left hospital September 12th, 1902	I diagnosed this case as one of acute appendicitis.
				C. Cases of Gastro-enterostomy.			
12	August 1st, 1902	M.	69	Painful dilated stomach, constant vomiting; tumour as large as a Tangerine orange at the pylorus; rapid emaciation	Gastro-jejunostomy by McGraw's elastic ligature	Complete recovery, with increase of weight and cure of dyspepsia	I thought the case was one of cancer of the pylorus; it was in reality pyloric and duodenal constriction due to adhesions resulting from the cicatrisation of an old duodenal ulcer or of inflammation round the gall-bladder.
13	March 10th, 1903	M.	50	Painful dilated stomach and thickened pylorus; constant vomiting; great emaciation	Pre-colic gastro-jejunostomy; direct suture	Complete recovery, with increase of weight and cure of dyspepsia; left the hospital April 22nd; seen in July, 1903	Fibrous thickening of pylorus, the result of swallowing 1½ ounce of strong nitric acid three months previously; there was no dysphagia.
14	May 20th, 1903	F.	30	Chronic and very painful dyspepsia with vomiting for last three years; much emaciation; stomach not dilated	Post-colic gastro-jejunostomy; direct suture	Complete recovery, with subsidence of symptoms; left the hospital July 2nd, 1903	Was very sick for many days after the operation; sickness at last overcome by champagne.
15	July 31st, 1903	F.	21	Chronic painful dyspepsia; admitted with the diagnosis of chronic gastric ulcer on the verge of perforation; almost constant vomiting	Post-colic gastro-jejunostomy; direct suture	Made a speedy and perfect recovery, and left hospital on September 5th, fourteen days after the operation	Felt so hungry seven days after the operation that she stole a currant bun from another patient; ate it, and afterwards passed the currants.
16	Sept. 4th, 1903	F.	58	Painful dilated stomach with much dyspepsia; very feeble; great emaciation	Post-colic gastro-jejunostomy; direct suture	Death on sixth day after operation	The union complete, and aperture sufficiently large close to pylorus; many adhesions round duodenum due to inflammation from gall-bladder associated with gall-stones.
17	Sept. 10th, 1903	M.	54	Painful dilated stomach with constant vomiting after food; a lump could be felt in region of pylorus; much emaciation	Post-colic gastro-jejunostomy; direct suture	Complete recovery, and left hospital on October 11th, 1903	Pain and vomiting absent since the operation; said he went out of hospital "another man."
18	Sept. 28th, 1903	F.	52	Cancer of the pyloric end of the stomach; no emaciation	Pre-colic gastro-enterostomy; direct suture; the whole of the pyloric portion of the stomach was cancerous, but the fundus was free from disease, though it was very thin and dilated; it was not possible to do a post-colic operation owing to adhesions	Death twenty-four hours later	The symptoms pointed to a suture having cut out at the point of anastomosis, as there was sudden collapse, though the patient had rallied well after operation.

Digitized by Google

ON SOME AURAL CASES.*

By STEPHEN PAGET, F.R.C.S.,
Surgeon to the West London Hospital; Surgeon to the Throat and Ear Department of the Middlesex Hospital.

It is worthy of note that our knowledge of the diseases of the ear and the throat, and our treatment of them, owed their advancement chiefly to the invention of proper instruments of examination. Seventy years ago there were no frontal mirrors, no otoscope, no Politzer's bag, and no laryngeal mirrors; and throat and ear cases were not much better off than they had been in the days of Ambroise Paré. We, if we want to study these cases, must be familiar with the best methods of examining them; and the right way is this—to examine, in every patient, everything: the larynx, the pharynx the anterior and posterior nares, and the membrana tympani of each ear. That is the quickest way of learning; just examine all the structures in each case till you are sure of recognising them in every case, and of knowing what changes in them are within the limits of health, and what are outside those limits. Eight or ten cases, taken in this methodical fashion, are worth twice that number half looked at.

Now, with regard to the instruments of examination, let me say one thing, to begin with, about the laryngoscope—that we must have everything in its proper place. The lamp must be just above the patient's shoulder, level with his ear; the laryngeal mirror must be all on his soft palate, and none on his hard palate; its shank must be rested against the corner of his mouth, his head must be well thrown back, and the surgeon must sit lower than the patient, so that the light from his frontal mirror is thrown upward on to his laryngeal mirror, not downward. We must begin by gettting the right attitude and the right relations, or we never see further than the epiglottis. As for the reversal of the laryngeal image in the mirror, the best way to study it is to draw the outline of the larynx on a bit of paper, and then look at it in a mirror.

The examination of the anterior nares is an easier task. Yet even here one mistake is not infrequently made; a slightly enlarged lower turbinate is mistaken for a polypus. This is an unpardonable mistake; and nobody will ever make it who has paid the least attention to the examination of the anterior nares, and has seen how the structures there, like the external features of our faces, are of different shapes and sizes in different people, all of them normal. And, after all, there is not much to see in the anterior nares; you see the septum and the lower turbinates, and sometimes you catch a glimpse of the middle turbinates, and that is all that you can see. And if you have not got a proper nasal speculum, you can make a good one out of a couple of bent hair-pins, tied together with a bit of elastic going round the back of the head.

The examination of the posterior nares is more difficult. The patient ought to lean his head a little forward and to breathe through his nose, keeping his mouth wide open. The tongue must be held down with a spatula, not drawn forward with a cloth. If you have not a rhinoscopic mirror you can make one out of a small laryngeal mirror, just bending the shank of it back and bending the mirror itself a little forward. It is worth the trouble of learning, to be able to see the posterior nares. For example, you can see adenoids, and thus obviate the digital examination; and I need not say that you are thereby exalted, in the minds of the child's parents, high above anybody who thrusts his finger up the back of the child's nose. Besides, the structures of this region are always worth looking at. The posterior border of the septum nasi is your landmark; find that first. You cannot mistake it, for it seems, in the mirror, to come toward you like the bows of a boat. Above it there is the vault of the pharynx; on either side of it there are the oval windows of the posterior nares, showing the grey and shining ends of the turbinates; and on either side of the posterior nares there are the large, round, prominent mouths of the Eustachian tubes, looking like the os uteri in miniature, one on each side.

Finally, we come to the examination of the ear. And I do advise you, very urgently, to learn the use of Brunton's otoscope. You will never get any pleasure out of looking at the membrana tympani unless you magnify this very small structure. It is no bigger than your little finger-nail, and you want to study it with the utmost care; every chance variation, and every pathological change. You might as well look at the fundus of

* From a Lecture delivered at the Polyclinic, October 2nd, 1903.

Digitized by Google

the eye without a lens as at the membrana tympani. Once make a habit of magnifying the membrane, and you begin to discover a whole host of things that you had overlooked; and the constant use of the otoscope not only improves your treatment of your patients, but adds immensely to the pleasure of your work.

What do we see when we examine the membrana tympani? We see a delicate lustrous fabric like the inside of an oyster-shell, slightly concave, and sloping a little downward. The handle of the malleus is woven into this fabric, just as the upright sticks in a basket are woven into the wickers. That is your landmark, the handle of the malleus; it crosses the field of the membrane, backward and downward, a thin little rod of bone with a knob like a pearl on the top of it. At the tip of the handle of the malleus, the membrane is drawn inward, and your light is caught here and is reflected back to you, just as it would be if you were to look through a speculum at the bowl of a silver salt-spoon. This is the bright spot, or what is called the cone of light: and, except that it shows that there is nothing wrong with this particular bit of the membrane, it has not the least importance of any kind. At the other end of the handle of the malleus, where the little knob comes (the processus brevis), the membrane is drawn into two folds or wrinkles, called the anterior and the posterior folds; but these folds vary a good deal, and their chief interest is that the posterior fold, if the membrane be retracted, is drawn tight and horizontal, so that it looks as though the malleus had two handles instead of one. Again, if the membrane be relaxed, or if it be bulged outward by fluid behind it, one fold or the other, or both, may be smoothed out and obliterated. But we are concerned at present with the normal membrane. If only you will always look at it through a lens, you will always enjoy looking at it: for it is one of the most elegant structures in the whole body, and we can only regret that such a fair feature should be hidden away at the bottom of a *cul de sac*. Besides, all the diseases of the middle ear are written, more or less legibly, on this scrap of membrane, both past and present diseases: and you may easily win a cheap reputation of wisdom by saying to some elderly patient, "I perceive that you had a discharge from the ear when you were a child." Indeed, the otoscope is to aural surgery what the stethoscope is to medicine, and it shows you all sorts of abnormalities and faults in the membrane which you will not see in any other way. For instance, if the posterior portion of the membrane be sunken or perforated, it may enable you to see something of the incus and the stapes, just glimmering through the veil of the membrane, or peeping through a hole in it.

So much for the examination of the membrane, and the aspect of it in health. Let me say something about some of those changed aspects, in disease or injury, which we most commonly see in the routine of aural work. There are more than we have time to consider to-day. Even if we left out the diseases, and took only the injuries of the membrane, there would be plenty to talk about. For example, there is that familiar picture, the transient congestion of the membrane after prolonged syringing. This hardly deserves to be called an injury, but the picture is worth remembering—the dilated capillaries running down from above, and the flush along the back of the handle of the malleus; it is nothing of any importance, and, indeed, these capillaries become dilated at the least provocation. Still it makes a very pretty picture: and the thing to remember is that this transient blush is due simply to the smack of the syringing on a sensitive membrane, and does not indicate any trouble behind the membrane. Then, for another example of injury to the membrane, there is the familiar picture of the membrane belonging to some foolish person who habitually picks his ears with matches, or hair-pins, or tooth-picks,—the little petechial hæmorrhages, scratches, or pin-pricks just scabbed over with a spot of blood, one or two of them on the walls of the meatus, and one or two on the membrane itself. Only last week, I counted seven of these scratches on a single membrane. Then there is the rupture of the membrane from a box on the ear, which is a single linear rent in the membrane, like a cut made with a blunt scalpel and scabbed over with dried blood; so that you cannot say whether it is or is not healed till you have made the patient test it by blowing air up his Eustachian tubes; but this test is likely to do more harm than good.

These injuries of the membrane, after all, explain themselves. Let us consider some of its diseases. Of itself, apart from the middle ear, it has no diseases worth thinking about; it is simply one

Digitized by Google

side of the middle ear, so placed and so constructed that we can guess by looking at it what is going on behind it. Take, to illustrate this point, two very familiar changes brought about in it by disease of the middle ear—retraction and relaxation.

Retraction of the membrane is best seen in children with adenoids, in whom these adenoids have caused obstruction of the Eustachian tube and stoppage of the supply of air to the middle ear. We need not ask whether the membrane is drawn inward by shrinkage of the tissues or is pushed inward by atmospheric pressure; whichever it may be, the picture of the retracted membrane can hardly be mistaken. The handle of the malleus of course goes with the membrane, and you see it foreshortened and almost horizontal; the processus brevis stands out very clearly, and looks as though it were on the point of jumping through the membrane; the posterior fold is drawn tight and straight; the spot of light is shifted, or broken up, or gone altogether; and there are dark shadows lying in front of the handle of the malleus and under the posterior fold. That is the ordinary look of the retracted membrane, sunken, but not adherent to the inner wall of the middle ear. The adherent membrane is, of course, a more serious affair. There is nothing more hopeless than a patch of membrane which has become stuck by cicatricial tissue to the inner wall of the middle ear, and, as it were, fused or soldered to it, so that you see the pink surface of the promontory shining through.

Take next the relaxed membrane, one of the clearest and most interesting pictures that you ever get in aural work. The whole membrane is not involved, save in very rare cases: but you see one bit of it which looks thin and bright and loose and crinkled, something like tissue-paper or fine gauze; and when the patient forces air up into the middle ear, this bit of the membrane is blown outward, just like a sail caught by the wind. Once you have seen this flapping forward of the membrane you can never mistake it—this sudden expansion and shifting nearer of a patch of membrane that was lying slack. And, of course, where the membrane is thus lax and without elasticity, you cannot expect to do good with Politzer's bag; the patient is already self-Politzerised and over-Politzerised, and you are likely to make things worse instead of better.

Let me now say a few words about three very common sets of cases which are worth thinking about. Forgive me that I speak at random of three subjects so far apart; they are (1) Otorrhœa in infants, (2) Cases that look like adenoids, (3) The significance of tinnitus.

Otorrhœa in Infants.

This, of course, is one of the commonplaces of an aural department; and the cases are all alike. The baby has a discharge from one or both ears, generally both. It began "of itself," was not preceded by pain, does not interfere with the baby's general health, is not markedly offensive or profuse, and is soon healed by very simple treatment. That is the ordinary picture of these cases, and they may seem to have no importance: but they raise questions of great interest.

First, it is to be noted that in every one of them there is middle-ear disease, with perforation of the membrane. We may be tempted to think, in this or that case, that the discharge is merely ceruminous, or due to some irritation of the meatus; but, so far as I know, this idea is wholly false. You have only to look at the ear and judge for yourself. It is true that we cannot be sure of seeing the membrane in these small babies, but we can be fairly sure of seeing the pulsation of the drop of discharge, or of the drop of lotion, lying at the bottom of the meatus; it just catches the light, and reflects it back to our eyes as a bright spot moving up and down, or from side to side, in time with the pulse. This dancing bubble lying in the hole in the membrane, and rising and falling like a float is a sure sign that the membrane is indeed perforated; and in small babies it is the only proof of perforation that we are likely to get.

Next, this otorrhœa of infancy reminds us of the fact that babies, even a few weeks old, may have adenoids. It is rare, but it does happen; you do find, once and again, a baby so blocked up with adenoids that it has great difficulty in taking the breast, and has to be fed with a teaspoon, and gets half-suffocated during sleep. And in such a case it may be right to operate, and necessary to operate, even a few weeks after birth. Short of operation, you may be able to do good by introducing a fine soft rubber tube the whole length of one or both sides of the nose: but in very bad

Digitized by Google

cases I doubt whether anything will take the place of the ordinary operation.

The mastoid in these cases seems always to escape; I have seen necrosis of the mastoid as early as three years old, but never, I think, in infancy. The discharge from the ear is usually stopped by very simple treatment: and the use of any strong antiseptic lotion is likely to do more harm than good. If you syringe a baby's ears with lotion containing carbolic or perchloride, they are likely to become sore; it is best to use nothing stronger than boric lotion.

Finally, comes the question whether this otorrhœa of infancy does any permanent injury to the hearing. It is so slight, and so easily cured, that we are in some danger of forgetting the possibility of lasting harm from it. We must promise nothing; we have no way of estimating, in these small infants, the extent of the harm that has been done. It may range from complete recovery to the very gravest loss of hearing.

Cases that look like Adenoids.

The longer one works in a throat and ear department, the more profoundly one is impressed by the vast number of children who, not having adenoids, yet show all the signs of them, and go through all, or nearly all, of the troubles associated with them. One sees, literally, hundreds of such cases—children whose looks and voices and histories seem to justify you in saying, "I will not distress the child with a digital examination; I will make the examination when the child is under the anæsthetic." Again and again, in every throat and ear department, children are sent up for the removal of adenoids who have not got any to be removed, or, at the most, have but a slightly thickened mucous membrane. What you do find in place of the expected mass of adenoids is a small, narrow, non-expanded pharyngeal vault; it feels as though the space behind the nose were crowded up and flattened out, hardly admitting your finger; the whole region is ill-shaped and ill-developed. And, of course, with this stunted post-nasal cavity there are the like narrowness and arrest of growth of all the lower part of the face.

What can we do for these cases that look like adenoids, and have all the troubles of adenoids? Indeed, it is not easy to do much for them. In some cases you must reduce the blocked and congested state of the nasal cavities; in some, but only a few, you may do good, as it were by chance, by curetting the naso-pharynx, even though you remove no mass of adenoids. But on the whole you will get more disappointment than satisfaction out of these cases, and the older they are the less you will improve them. Perhaps the galvano-cautery, in the long run, is the most effective treatment for the older patients. The point is that we must recognise this fact—that the multitude of children who look like adenoids, but have not adenoids, is not much less vast than the multitude of children who have adenoids.

Let me add here one hint how to examine all these cases. First, if the patient be a child, take him on your knee and support his head comfortably with your left arm; do not attempt to examine him while he sits on a chair. Next, to get your finger well up behind the palate, keep away from the uvula, away from the middle line, and keep close to one side of the throat; there is always room just above the tonsil for the tip of your finger.

The Significance of Tinnitus.

Sir William Wilde used to say of tinnitus that they who were subject to it heard those sounds that were most familiar to them: the country people heard birds singing, or water running; the town people heard noises like hammering and sawing and the traffic of the streets; the old women heard tea-kettles boiling, etc. I do not think that you will get any help from this theory; but I do think that you will find it worth while to draw a line between those sounds that are simple and those that are complex. One patient will complain of a mere hissing, buzzing, or humming; another will give a vivid and accurate description of some complex, articulate, elaborate, picturesque group of sounds. "It is just like a lot of birds singing in the trees," they say; or, "It is just like so many church bells ringing all at once, some quite close and some a long way off." *Just like* this, *just like* that; they describe it all with the strangest preciseness of words. Be sure that these cases of complex tinnitus are the hardest to relieve, and are all, or nearly all, past cure; the more articulate, more harmonious, more elaborate sounds are evidence that something is wrong, not only with the conducting apparatus but with the perceiving

Digitized by Google

apparatus; and I do not see where we can draw a line between these cases and the cases of auditory hallucinations. Take, by way of commentary, the following case:—A naval officer, æt. 38, came to me complaining that he heard voices. At night he would hear them just as he was dropping off to sleep, or he would be waked by them; and the sense of spoken words was so strong that he had even got out of bed, believing that somebody was about the house. I do not deny that his ears showed signs of past injuries. One membrana tympani was relaxed, the other was slightly congested; and he said that he had suffered from fever in Africa, from many attacks of influenza, from heavy gun firing, from sea-bathing, and from impacted cerumen. But practically his hearing was perfect; he had no vertigo: and his tinnitus was extraordinarily complex. This sense of clearly spoken words, heard halfway between waking and sleeping, is a very strange thing; it may last for years, and then get well of itself; it is more like an aura than it is like a tinnitus; and, in the case of this particular patient, though it was perhaps started by some kind of middle-ear derangement, yet it was of itself a central trouble, to be treated not by Politzerising, but by attention to his general health. This, of course, is an extreme case, but it may serve to illustrate the point which I am trying to make—that the significance of tinnitus is to be measured by the complexity and elaborateness of the sounds heard. A plug of wax may suffice to cause a "buzzing in the ears;" but if a patient describes a whole series of complicated intelligible sounds—songs of birds, pealing of bells, clanking of chains, and even sounds of human voices,—be sure that you will not do much for him by any of the routine methods of aural surgery.

THE statement of Bard that the orbicularis sign is invariably lacking in peripheral facial paralysis, and is therefore a distinguishing mark of central affection, is incorrect. The presence of the sign in peripheral paralysis is further proof of the existence of commissural fibres between the nuclei of the seventh nerves. The sign is of clinical value in so far as its presence in peripheral paralysis shows that complete recovery has not yet occurred. The secondary over-action in the orbicularis palpebrarum does not occur in those cases in which the sign, having been present, has passed away.—*Journ. A. M. A.*, Nov. 7th, 1903.

IS AN ECZEMA EVER SYMPTOMATIC?

By T. ROBINSON, M.D.,

Physician to the Western Skin Hospital.

THE varied aspect which an eczematous inflammation presents will be familiar to every medical man. So varied, indeed, is the condition of the skin which is designated eczema, that it would be far better if we dropped the substantive and used the word only as an adjective. Eczematous inflammation of the skin has markedly three characteristics: itching, infiltration, and persistence, and we might add a tendency to recur. Our difficulty in studying eczema has been increased by admitting the doctrine that some unknown condition of the tissues gives rise to an inflammation in a certain spot, which inflammation has all the characteristics of eczema. What this state is no man knows. Treatment being based upon this assumption—for it really is nothing more,—patients are too often drenched with drugs.

The laity cling pertinaciously to the blood origin of all skin diseases. They speak of the blood being impure, or "out of order," or "too hot," which indicates a popular belief in the theory that skin diseases, especially eczema, are only symptoms of a pre-existing morbid state of the blood. These pre-existing or existing morbid conditions would be represented by tuberculosis, diabetes, and gout.

If we reflect as to the characteristics of tubercular conditions, we shall, I think, find that they have three distinctions: they are slow, low, and not irritable. Tubercular lupus will bring before our mind a special example. The clinical error made in associating eczema with tubercular disease arises probably from the fact that the victims of consumption are individuals with faulty and vulnerable skins, and that their tissues are easily disturbed and drift into an eczematous condition; but we are firmly of the opinion that the tubercle bacilli does not play any part in eczematous inflammation.

In cases of diabetes we do find pruritus vulvæ, which is an eczematous inflammation occurring on a tissue which represents the transition from skin to mucous membrane, and is caused only by the saccharine secretion trickling over the surfaces adjacent to the urethra, and cannot by any process

Digitized by Google

of reasoning be called symptomatic of diabetes. We are not acquainted with any other association between an eczematous inflammation and diabetes.

Does the presence of uric acid or its compounds ever initiate an eczema? Certainly it is true that quite a number of those who are eczematous have, at the time of our seeing them, some evidence of gout, or they relate to us testimony which proves they have the gouty diathesis. The characteristics of all gouty inflammations are threefold: they are sharp, sudden, and fugitive. Eczema may be sudden in its onset, but it is not a fugitive condition. Direct investigation in scraping the secretion from any eczematous area and examining the same for urate of soda or uric acid has given negative results. There is one other argument against eczema being symptomatic of gout, which is that the administration of colchicum in cases of eczema does not act as a curative, and, so far as has been observed, is without any effect. The common association of gout and eczema being an admitted clinical fact, we must look for an explanation other than the belief that eczema is actually lit up by the irritating effect of urate of soda or uric acid. Is there such an explanation? Let us face the difficulty. It will be found that the patients who suffer from eczema have an imperfect skin—imperfect, that is, in being physiologically ill-balanced, either sweating too much or too little,—and it is among these whose power of elimination is inadequate that we venture to think we find the association of gout and eczema. The skin being the great ejector of effete matter, and the action being insufficient, a greater amount of work is thrown upon the kidneys, and probably lungs, than they are capable of performing; hence the accumulation of the urea in the blood, and in the course of time the development of gout.

If we can accept the origin of an eczema as being always external, we shall approach the study of the causation of the disease with greater ease and greater freedom, and, I may add, with more hope of success in treatment. If I turn to my case-book I find the last three cases of eczema easily traceable to external causes. One, an officer in the army, had a patch of eczema over the ball of his right thumb which corresponded to the pressure caused by the hilt of his sword. Another was an eczema limited to the brows, and was apparently caused by the pressure of a high hat. My third example was a patch of eczema over the radius, apparently caused by the friction of the shirt-cuff, which rubbed the exact position where the eruption was. How simple a matter to take away the cause in each of these cases for the production of relief, and how futile to hunt for any blood state or to give internal remedies. A complete revolution is taking place in our way of looking at all disease, and as time goes on, and we trust to our own powers of observation, I firmly believe it will be found that few, if any, diseases are born in the organism. Senile pathological conditions will, of course, be found due to natural and inevitable degeneration. The law of parasitism will assist us more in understanding the nature of disease than all the chemistry and physiology which has ever been written. We must be prepared in the future to trust more to the knowledge which will go into our brains through what Bunyan called the gates of the body; and of all gates the eye-gate is the most important to a doctor. We must not lean too much upon investigation crutches. Nothing can take the place of clinical observation; there is so much to learn in any simple malady such as eczema, so much which is not clear, that we shall find ample field for our energies and our abilities. We must look for the common, easily seen characters of the malady. We must, in other words, use a shovel and not a teaspoon; if we do not we shall miss a great deal which will help us to understand and which will assist our patients in gaining health.

Paraffin Prothesis.—Morton reports a number of cases, and illustrates his views with microscopic drawings showing that the paraffin disappears and is replaced by connective tissue and embryonic tissue within four months. The dangers of sepsis, emboli, and sloughing are mentioned. Sepsis can be prevented by asepsis; emboli by using the paraffin at the proper temperature, though experiments seem to show that paraffin, the melting point of which is below the temperature of the body, gives rise to thrombosis. He doubts whether this is as dangerous as the experiments would lead one to believe. He uses paraffin of a melting point of 109° F., brought to this point by adding sperm oil, and sterilised by placing in a water bath, and exposed to heat sufficient to boil the paraffin.—*Journ. A. M. A.*, Nov. 7th, 1903.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 580. WEDNESDAY, DECEMBER 9, 1903. Vol. XXIII. No. 8.

CONTENTS.

PAGE

*A Clinical Lecture on Varieties of Arthritis. By SIR DYCE DUCKWORTH, M.D., F.R.C.P. 113

*With Dr. Campbell Thomson at Middlesex Hospital—General Paralysis of the Insane 119

*A Clinical Lecture on Itching as a Symptom, and its Treatment. By G. NORMAN MEACHEN, M.D., B.S., M.R.C.P. 121

Reviews of Books 128

** Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

ON

VARIETIES OF ARTHRITIS.

Delivered at St. Bartholomew's Hospital.

By Sir DYCE DUCKWORTH, M.D., F.R.C.P.,

Physician to the Hospital.

GENTLEMEN,—I want to speak to you to-day about the subject of arthritis. You will notice that I use the term in its simplest form. Arthritis means an inflammation of the joints. As a matter of fact there are many forms of arthritis; for instance, there is osteo-arthritis, gouty arthritis, septic arthritis, and so on. In speaking of the genus arthritis to-day, I hope to illustrate it by several examples, as we have at the present time a rather large series of cases of the condition in the wards. Inasmuch as clinical lectures differ from systematic lectures in that they relate to the actual presence of the patient and his present condition, I am always glad to illustrate the topics of the lectures by the presence of the subjects of the condition themselves. The patients who are here are not presented exactly in the order I expected and hoped, but we will deal with them first, as they cannot stay very long. The first case I will refer to is that of this female patient, who has been in the wards a long time and has been suffering from an arthritis which, in her case, is only a manifestation of the general disorder from which she suffers. She came in last November, and from that date till now, some seven months, she has been in a feverish condition, as will be seen by the charts I shall hand round to you; it has been a very chronic, long-continued pyrexia. After studying those charts you will be surprised to find the patient in such a satisfactory condition as she is, for her temperature often ranged as high as 103° and 104°. Her complaint is an affection of the joints which you can see by her wrists; they are very much swollen and knotty.

Digitized by Google

It is symmetrical in both wrists. It is very painful sometimes; at others less so. You will notice also that the elbows are greatly swollen, and the muscles of the forearm and upper arm considerably wasted. The shoulders are sometimes much involved in the disease, and occasionally the knees also. Therefore this is a case in which many joints are affected, and to that the name polyarthritis has been applied. I have told you that the joint symptoms here constitute only a part of this particular case. Both knees are enlarged and swollen on account of the presence of effusion in them, so that the patella floats and can be pressed down in the intercondyloid space. Her ankles have been painful too. The hip-joints appear to have escaped. There are some other remarkable features about the case, which I will speak of afterwards. She did not come here on account of the state of her joints; she was sent up from the country suffering from a febrile condition, and then she was found to have an enlargement of the spleen. Since last November we have made a prolonged study of her case, which has enabled us to come to a clear opinion as to the nature of it. It is one of infective endocarditis. She has a murmur in the area of the mitral valve, indicating endocarditis, and that is the source of the evil in her case, infective endocarditis being a form of chronic pyæmia, or, as it has been called by Sir Samuel Wilks, arterial pyæmia. Her joint trouble has certainly been the most painful part of her illness. As a rule, when the joints become more painful there is an increase in the febrile condition. After a day or two of the great pain it subsides, and then the temperature also goes down somewhat. It is noteworthy that she has had no attacks of hyperpyrexia, there have been no flights up to 105° or 106°, and there have been no rigors, but the pyrexia smoulders on and the condition of arthritis continues.

We will now take the next case, which is also that of a female. This patient has frequented the hospital since 1890—that is to say, thirteen years ago,—and this is the fourth time she has been in suffering from the complaint. When she suffers severely she returns to the hospital. You will notice she is very thin and poorly nourished. Her complaint from the first has been with regard to her joints. I ask you to observe her hands, which are very characteristic and typical: for after you have once seen such hands you will be likely to remember them when you meet with other cases of the kind. Her prominent conditions are swelling and enlargement of every joint, with projections or exostoses at the end of the ulna, enlargements of the metacarpo-phalangeal joints, the phalangeal joints, with effusion into some of them. There is even enlargement of the extreme distal joints. Together with that there is generally a very moist and clammy condition of the hand—a condition which may be called cold and frog-like—and combined with that there is a general wasting of the muscles of the part, including the interossei and thenar and hypothenar muscles. There is also a little permanent flexion, because the tendons have become drawn up. At the elbows the ends of the bones are enlarged and both condyles are prominent. Both her knees are severely involved. She is unable to straighten her arms completely. This is a typical example of osteo-arthritis, or rheumatoid arthritis, as it was called by Sir Alfred Garrod in 1858, a name by which it is very generally known, though there are many others. One of the commonest names is rheumatic gout, and it is one of the worst which can be given to it. Other names are nodular rheumatism and dry rheumatism. Most of these patients present a condition of poor health, great wasting of muscles, and any joint may be affected, even the jaw or the spinal joints. These latter forms are, happily, very rare, and you can imagine they are trying cases and melancholy to witness. This patient is in a bad state, but I do not think she will become worse. Her greatest pain at the present time is in one knee, which is very much swollen; there is an obvious enlargement of the end of the femur and the upper part of the tibia. The patella seems flattened out. There is great thickening about the surrounding parts. For the last four or five years she has been fairly comfortable and able to do her work, which is that of a book-keeper; but she is having another relapse and has come back to us. It is very difficult to be dogmatic as to the etiology of this case. There is no history of rheumatic fever, so it is not the outcome of that disease. I show you the negative of a skiagram of her hands, which shows very well the enlargement of her bones; the enlargements of her phalangeal bones show little exostoses. Often there is also some hypertrophy of cartilage, and there may be effusion from time to time into the capsule of these

Digitized by Google

joints. In many of these cases a curious deformity is noticed—namely, deflection of the fingers towards the ulnar side, a symmetrical deflection. The metacarpal bones are quite straight, but the phalanges go off at an angle towards the ulnar side of the body. They constitute the "seal-flapper" type of deformity. That is not peculiar to this form of arthritis; it is found in other forms of arthritis, provided the condition has been of long standing. In course of time the digits are brought to the ulnar side by muscular action. In this case, although the condition has gone on so long, she has not got this seal-flapper type of deformity.

In the next case we take up another form of arthritis, in a male patient well advanced in life. He is fifty years of age, and came in on April 3rd last. He had been ailing for twenty-seven years—that is to say, more than half his life,—and he has been affected with this form of arthritis since he was twenty-three years of age. The first joint affected was that of the great toe, and since then he has not passed a year without one or sometimes more attacks in different joints. Almost all the joints of his body have suffered in turn. The attacks come on very suddenly, and there is pain and swelling and redness. After the attack has passed away the patient feels much better, but the joints appear to be crippled a good deal after the attacks. Sixteen years ago he found that certain deposits were being formed upon the back of his hand, and since then large masses have formed upon the hands, feet, and elbows. Sometimes also these deposits have occurred on his ears. Many of you will recognise this form of arthritis as one which is familiarly known by the name of gout. There is no doubt about the diagnosis in this case; it is gouty arthritis affecting many joints, and therefore it is a polyarthritis. It has also received the name polyarthritis uratica. Here, then, is another etiology for arthritis. Some photographs have been taken of this man's hands, and you will see that extraordinary deformities have arisen in connection with these deposits. One remarkable feature about this man's case is that he lived about thirty years in New Zealand, one of our colonies where the people are very healthy, robust, and industrious, leading such lives as are commonly supposed to prevent gout. It has been said that if a man can earn sixpence a day and live upon it he will never be gouty, and I think perhaps that is true, for it goes to indicate activity of body without any grossness of living or feeding. This man's condition is accompanied by extraordinary deposits commonly known by the name of chalk-stones. But it is not really chalk, for that is carbonate of calcium, whereas this is urate of sodium, with small quantities of lime salts in it, which have become deposited in great quantity all round the digits. These masses lead to wasting of the skin, which becomes exceedingly smooth and soft, so that you can see the deposit working its way peripherally by centrifrugal pressure. Not only are the joints affected, but the textures and tissues around are infiltrated with this sodium urate, which is apt to be deposited when the circulation is slow and sluggish. You will see one deposit in the helix of the ear; from time to time these deposits in the concha grow out, and they can be taken away like little pearls. I have seen these deposits on the nose, in streaks down the thigh, and on the dorsum of the penis. Sydenham, 250 years ago, described them, and called them "tartar of the blood." Just as cream of tartar is deposited in a cask of wine, so does this particular deposit settle down out of the blood. The feet are involved very much in the same way. Here, for instance, is an enormous mass over the tuberosity of the tibia. Probably that has been a bursal sac, which has now become stuffed full of urates. The whole joint is encrusted and infiltrated with these chalky deposits, and gout is the English name for it. Sometimes this deposit takes place without any obvious classical attacks of the disease. I mean that there is no pain, so that the patient may be infiltrated with urate of soda without ever being aware of having had ordinary gout in his life. This has ultimately crippled the patient. Generally, the deposits occur more in the upper extremities than in the lower, the hands being affected more than the feet. This man is peculiar in that he never appears to have done anything to deserve his gout; he has not been a gross feeder or a hard drinker. In New Zealand the people mostly drink whisky, and not much beer or malt liquor. There are several reasons for that. I suppose the majority of New Zealand colonists are of Scottish origin, and they keep up the use of their national beverage. It is the case also in Canada and in most of the colonies, with the exception of

Digitized by Google

South Australia, where I daresay a good deal of beer is consumed. Whisky drinking does not produce gout in Scotland; I suppose there is not a Scottish peasant from the Tweed to John o' Groat's who has had an attack of true gout, and I doubt if you could find two in the whisky-drinking peasantry of all Ireland. Porter has, however, become largely employed in Ireland, and before long we may learn that gout is beginning to appear there amongst the lower orders. Gout occurs among the English specially; there is more gout in England than there is in any other part of the Empire. But there was some gout in this man's ancestry; he had a gouty uncle. Therefore the tendency is in his constitution, and in his race. That is the peculiarity of gout; it is a personal matter, one inherits it along with one's tissues and tendencies. The metabolism of the gouty man is not that of an ordinary perfectly sound healthy man; the metamorphoses of his system go on in a different way from those of other individuals, and he creates toxins in the process; thus the poison of a gouty person is that which he makes himself. That we believe to be a very different form of toxin to that which induces rheumatic attacks. The rheumatic patient is poisoned also, but he does not make his own poison; it is infective, and comes from without; it is an invasion of toxin from without, like that of smallpox, scarlet fever, or measles. This is a very crippling form of arthritis, because the chalky deposits burst through the skin and discharge a pasty matter like white paint, after which relief is felt in the part. Now you must not be tempted to employ surgical interference in these cases. Operations upon tophi by enterprising surgeons have been followed by erysipelas and cellulitis, and I have seen other serious results. When the knife is put into them you do not find that the urate comes out in the rapid way you might expect; it is removed by a slow exudation, it takes its own time, though you may now and then assist by putting in a probe so as to enlarge the opening and employ gentle pressure. Here I show you a skiagram of this man's hand, showing how it is involved. You will notice the deflection of the phalanges and enlargement of the ends of the bones, just as in rheumatism. It is certain that uratic arthritis may deform the bones in almost precisely the same way as the rheumatic poison. Here I show you a specimen of a gouty joint in which there is cartilage encrusted with urate of soda, just as if one had treated it with white paint. It is about eighty years old, and it was prepared by Mr. Stanley for the museum of this hospital, and it is still as good as on the day it was put up. In those days the museum preparations were preserved in pure spirit of wine. In later times, since spirit of wine became contaminated with methylated spirit for the sake of cheapness, it was found that when gouty joints were put into methylated spirit, that preservative began to exercise a solvent action upon the urates and dissolved them out, so that the preparations ceased to be of any interest. Now, therefore, we are careful to put all gouty preparations into pure spirit of wine. A large series of preparations by Dr. Norman Moore were lost within a year or two after they were put up because the urates were dissolved out, which caused them to look like specimens of rheumatic disease. That showed that many of the lesions in the joints were exactly those of chronic rheumatoid arthritis. Having had the urates dissolved out of them, there remained the damaged joints showing the same conditions as presented by chronic rheumatic disease, *minus* the urates; in fact, the effects upon the joints are very largely the same in both cases, so it is small wonder that there should be a difficulty during life in determining which cases are truly gouty and which are truly rheumatic.

We have had here to-day three instances of arthritis: one where it has occurred as part of a septic process in the course of infective endocarditis; another a perfectly typical case of rheumatoid arthritis in an exhausted and ill-nourished young woman, where it has been exceedingly chronic, having gone on for thirteen years and still having acute phases; and another an illustration of arthritis due to gout in a very accentuated form associated with large tophi. Here is a specimen showing a good illustration of a lesion produced in chronic rheumatoid arthritis by friction of the patella, which is polished in such a way as to make it look like ivory, and you can see ledges there where the erosion has taken place. Outside you get the process which is so well seen at the end of the femur, called lipping, a sort of overflow, as if the joint had been fused and by pressure a ridge had been formed. That lipping is one of the most important features in a case to pay attention to. It is the

Digitized by Google

site of greatest pain. That is true both in gout and in rheumatism. There are other conditions which lead to arthritis which I shall only have time to enumerate. One of the commonest is the poison of gonorrhœa, which comes into the septic category. It is called gonorrhœal arthritis; and some people call it gonorrhœal rheumatism, which is a bad name. Do not use the word rheumatism except in cases where you believe the condition to be truly infective and rheumatic. With poor people, and even well-to-do people also, if they have a pain anywhere it is called by them "rheumatics," so that rheumatism in such circumstances may be simply regarded as another name for pain; and it is your business to find out what the particular cause of the pain is—whether it is septic, or rheumatic, or the result of an injury. It is a curious fact, and one which should be borne in mind, that people who are of a gouty habit of body are more vulnerable than other people to the gonorrhœal poison; if a gouty person contracts gonorrhœa he is sure to suffer from it more than anybody who is not gouty, and it will linger longer in his system. It is also very likely to set up such troubles as iritis and conjunctivitis, and above all it is likely to set up arthritis in one or more joints, generally in one, and its result may be a crippling of the patient for life and the ruin of his whole career. Another form of arthritis is recognised as due to the influence of the nervous system. It is called neural arthritis or neuro-arthritis. I think it was first found during the great American Civil War that many of the gunshot injuries of the spine and spinal cord led in process of time to curious forms of arthritis and swelling in the joints, and it is now well recognised that certain injuries may be accompanied by neural arthritis. It is certain that locomotor ataxia, a disease affecting certain columns of the spinal cord, is accompanied by arthritis, and what is called Charcot's disease is an extraordinarily acute form of arthritis arising in one or more joints, so that in a few days the joint becomes distended with fluid, what is called a hydrarthrosis, all its constituents breaking up and producing a huge knee-joint which may swell up to the size of a child's head, the bones being separated and the limb hanging loose like a flail. It is a riotous raging arthritis occurring in the course of this spinal disease. Here there is no question of septic matter, there is no question of rheumatism as ordinarily understood, nor is there any question of gout; the disease is believed to occur under the influence of damage to the spinal marrow. Traumatic arthritis is readily understood; it is a direct injury to a joint which leads to inflammatory changes in all its component structures. But there is this to be said in respect of traumatic arthritis,—that an injury received by a joint in a person of certain habits of body will often betray that habit of body which he possesses. For instance, if a delicate child receives an injury to its knee or any other joint, that joint becomes swollen and painful and does not get well under ordinary rest and proper treatment; a lingering process goes on, formerly known as a white swelling. In other words, there is a chronic arthritis produced by the injury in a sickly child of scrofulus or strumous habit, and that form of the disease is set up which is now known among surgeons as tuberculous joints. Probably they are tuberculous by the time the surgeon sees them, but there is a stage of delicacy or vulnerability preceding that, as I have always maintained, before tubercle appeared, which is liable to be readily irritated. Such a joint is very apt indeed to suffer from tubercular invasion. Or again, if a gentleman who practically eats two dinners a day and drinks half a bottle of port, goes out riding and gets an injury, such as crushing of a limb against a tree, he will probably soon have a severe attack of gout in the joint or part which has been injured. As a matter of fact the injury was the touchstone for the gout, for the man was in an explosive state, and the injury, as it were, provided the match. So the habit of body is illustrated and manifested, just as in the scrofulous child the condition was not brought out until the injury occurred. So you see what a complicated subject is that of arthritis as a whole. Although we know more about it than our forefathers did, there still remains very much more to be known. It is certain that many varieties of arthritis are due to sepsis. That was so in the first patient you saw, the case of infective endocarditis. We have reason to believe that her condition is due to poisoning by streptococci. We have treated her with antistreptococcic serum, but so far without benefit. Bacteriologists tell us there are many varieties or strains of streptococci, and that the results produced may vary with the particular form which induces the disorder in each

Digitized by Google

case. We try to discover the variety of streptococcus in each patient, and for that purpose we take blood and have it cultivated, or we withdraw some of the effusion from the joints and cultivate that, so that we may ascertain what is the particular form of peccant material which is producing the disease. We have done that in this case, and the materials were sent to the laboratory and tested by experts, as you know. But the report was that in each instance they proved sterile. All we can do is to inject a mixed antistreptococcic serum into the patient, in the hope that whatever form is causing the mischief may be neutralised by part of the mixture. That sometimes is efficacious. For instance, last year we saved a boy's life by this means. The rule in these bad cases of streptococcal infection is that the patients die. One case under my care lasted over a year. This poor woman has been in since last autumn; she seems to be of a wiry constitution and bears this chronic fever very well; she has got wretchedly thin. Nothing I have yet given her has been instrumental in bringing the process to an end. Probably the source of poisoning in her case is vegetations on the cardiac valves, which from time to time discharge peccant material into the blood-stream, causing this fever and wearing her out. We have tried those drugs which are said to combat this condition, more especially the salicylates, the class of medicine which is most useful for all forms of arthritis; there is hardly any form of arthritis in which salicylates are not useful. It is most useful for conditions which are most nearly allied to rheumatism. Very often the drug is not given in large enough doses, so in this case we have pushed it as far as it is safe to do so. As a rule the subjects of rheumatoid arthritis are poor broken-down creatures, and it is necessary to feed them well, which is quite the reverse of what you would do in the case of a gouty patient. We give these patients stout, meat, and other nutritious food. A very good salicylate preparation is aspirin, an acetyl-salicylic acid. That has often produced favourable results in the matter of lowering of temperature, and it has been so in this case. There is a very lingering case of gout without chalk-stones in Matthew Ward, a case of polyarthritis uratica. The patient has been in before on one occasion, and was then the victim of gonorrhœal arthritis, and that proved his gouty habit of body, as did other things. He has been a very dissipated man and has drunk a good deal. The gout affects many of his joints, and it is of the form which generally afflicts those who have been habitually intemperate both in the matter of liquors and in venereal excesses. In such cases the gout is apt to be more troublesome than in ordinary cases. We are not aware that there is any gonorrhœal influence now in the case. His temperature has kept up persistently, and he has been unaffected by full doses of colchicum or salicylate of soda. But his temperature has been controlled under the influence of aspirin, and he is now having it in fifteen-grain doses three times a day.

Any source of pyæmia, whatever it may be, is a possible source of arthritis, and therefore a single carious tooth may be enough to set up arthritis. No doubt it does so oftener than is generally thought. When you get patients, especially young patients, the subjects of arthritis, it is a good thing to look out for carious teeth, which are often found to be the cause of the mischief. In view of the large number of cases which may set up arthritis, you have to ask yourself in any given case, what has set this up? There are many instances of disease which you will not be likely to treat successfully unless you find out their cause. There is a great deal which might be said in regard to the details of treatment which each class of case severally requires, and some of these points I have discussed elsewhere. I invite you to inspect these specimens of arthritis from the museum. One shows an extreme overgrowth of cartilage and of the synovial membrane; and another shows lipping of the ends of the bones of the phalanges. One of the best things to do with these cases of rheumatoid arthritis, especially in young people, is to send them away to a safe climate. My own opinion is, you cannot reach what I call a safe climate until you travel one or two hundred miles south of Cairo. If you can send your patient to India during the cold season, it will be found very suitable for osteo-arthritis. Certainly patients who are able to go to Assouan are found to benefit enormously. It is a very expensive treatment, but is is well worth the trouble and expense for those favoured few patients who can afford to travel so far.

Digitized by Google

WITH DR. CAMPBELL THOMSON AT MIDDLESEX HOSPITAL.

CLINICAL STUDIES IN DISEASES OF THE NERVOUS SYSTEM.*

GENERAL PARALYSIS OF THE INSANE.

GENTLEMEN,—This patient, a man æt. 29, complains of nervousness which, he says, he has had for three months. When questioned more particularly he is, as you see, unable to define his symptoms in definite terms. He states positively that he has never had syphilis. So far, there are no special points in the history to guide us to an exact diagnosis, but you will have noticed, while we have been talking to him, a peculiarity in his speech, and also a tremor of the lips when he talks. The speech is a hesitating one; the different syllables of words are often run into one another, so that it is difficult to understand what he says. This speech and tremor are suggestive of general paralysis, and we will now examine the patient to see if there are any other signs to confirm this suspicion.

The pupils are unequal, the left being larger than the right; both react to light but sluggishly, and they also contract during accommodation. Words of several syllables cannot be pronounced plainly; there is a marked tremor of the lips when he speaks, and of the tongue when it is protruded; there is also a tendency to over-action of the muscles of the face. There is a fine tremor of the hands; the knee-jerks are both greatly exaggerated, and the deep reflexes of the arm are increased. The plantar reflexes are present and of the flexor type, and there is no ankle-clonus.

These signs, then, taken together, are sufficient to enable us to diagnose this case as one of general paralysis. It is often difficult to know on how few signs one is justified in diagnosing any given disease, but in this case the symptoms taken collectively do not leave any room for doubt.

It is unfortunate that the patient's wife has not come with him, for it might then have been possible to have obtained some reliable information about his mental condition; for, as you know, mental changes as shown by loss of memory, carelessness in business matters, errors of judgment, and extravagance, are frequently among early symptoms of the disease. On questioning the patient we are not able to obtain any definite information on this point, for he says he is able to work as usual, but acknowledges that his wife has remarked the change in his speech, although he has not noticed it himself.

In the early stages this disease may bear some resemblance to disseminated sclerosis, the chief symptoms common to both being tremors, alteration in speech, and increase of deep reflexes; but it must be remembered that the knee-jerks are by no means always increased in general paralysis—they may be diminished, unequal, or absent. The tremor of general paralysis is a much finer one than that of disseminated sclerosis, and it is, moreover, especially marked in the tongue and lips. The speech, too, when well developed, is very different in the two cases. In general paralysis the speech is hesitating and syllables are dropped or telescoped into one another, while in disseminated sclerosis it is jerky, staccato, or scanning—characters which are rather difficult to describe, but which are distinctive when heard. Among other conditions which may simulate general paralysis are some phases of mental disorder due to chronic alcoholism, and some cases of syphilitic (probably gummatous) affections of the brain.

In the early stages care must be taken to avoid confusion with neurasthenia. Occasionally it may be impossible for a time to make a certain diagnosis, but usually there is a considerable difference between the simple fatigue symptoms of neurasthenia and the mental degeneration of general paralysis; and with the development of physical signs the differences become clear. In the neurasthenic who came into the room just before this patient, we noted how he worried about himself and his work, and how anxious he was to explain fully every one of his symptoms; while, on the other hand, the general paralytic whom we have just been considering is contented with himself, and in no wise inclined to magnify his slighter ailments. The neurasthenic worries about himself, but in general paralysis it is the friends who are often first worried on account of the mental symptoms, which change the man from his former

* These "Studies" are from the teaching in the Out-patient Department of Middlesex Hospital.

Digitized by Google

self into an irritable, careless, and otherwise strange being. The prognosis is bad, as it is with all cases of this disease. The patients, with the exceptions of occasional curious remissions, tend to go steadily downhill. Convulsive fits, epileptiform or apoplectiform in character, are often a marked feature, and each one leaves the patient much worse than before. As the mind deteriorates the physical weakness increases, until at last the patient arrives at that unhappy state which justifies the use of the term "general paralysis."

You will remember that this patient stated emphatically that he had never had syphilis. This is an important point, because syphilis is now generally thought to be the great underlying factor in general paralysis, as it is in locomotor ataxy. It, of course, by no means follows that when a patient denies having had syphilis he may not have had it, for there are many cases where the primary symptoms pass unnoticed, and attention is only drawn to it by the onset of undoubted tertiary effects; but even then the fact still remains that there is a considerable percentage of cases, both of locomotor ataxy and general paralysis, in which there is no reason to suppose there has been previous syphilis. This, of course, in no way lessens the importance of syphilis, but merely shows that there may be other conditions under which the nerve-fibres will also degenerate.

Syphilis apparently acts by lowering the vitality of the nerve-fibres in such a way as to predispose to their premature death. We thus see how it is that, with the vitality of the fibres lowered, other factors, such as worry, alcohol, illness, or injury, may act as exciting causes. The fibres, their vitality already enfeebled, might still just be able to live under ordinary circumstances, but wither and die under the influence of extra stress.

Lastly, we come to the pathological changes underlying this interesting disease. To the naked eye atrophy of the cerebral convolutions, especially those of the frontal lobes, together with thickened and adherent membranes, are among the principal changes. Microscopically, degeneration of bands of fibres connecting various parts of the cortex with one another can be demonstrated, and chemically it has been shown that when the degeneration has begun certain chemical substances are formed as products of the degeneration, and these act as poisons to the still healthy fibres, and thus a circle of events is established which ultimately ends in complete physical and mental wreck.

To-day, gentlemen, the patient's wife has come up to the hospital with him, and from her we have been able to gather some more important facts in connection with the case. His wife tells us that she has noticed changes in her husband for the past twelve months, which, you see, is a much longer time than the three months that the patient mentioned last time. During this past twelve months some alteration in speech has been noticed, and this alteration has been more marked lately. We are told also that the patient has altered mentally. He is irritable, forgetful, and inclined to be depressed—three important changes very common in this disease. His power for work has suffered, for, although his occupation is apparently simple and requires no special skill, he cannot do so much as formerly.

Early mental changes vary in character with different individuals; in some cases depression is a feature, while in others exaltation is present and may be accompanied by extravagance and recklessness, with definite delusions of wealth and grandeur. Just as some people become gay and others depressed when under the influence of alcohol, so it is in general paralysis: some are unduly elated when their higher nerve-centres degenerate, while others are proportionately depressed. Forgetfulness is especially apt to be noticed as an early symptom when the patient is in an important business, for naturally any mistakes are soon liable to be found out; but where the patient has not a responsible position, loss of memory, although perhaps present, would be more likely to be overlooked.

It is the same with manual work. In the case before us the work has not required skill; therefore the only change noticed is that the work is done more slowly than it used to be. Had it been highly skilled work we should have a decrease in quality as well as in quantity, for it is always the highest and latest acquirements that are the first to go on account of the failure of attention and delicate co-ordination which are required for their performance.

This patient is a married man aged forty-two. He complains of vague head symptoms. He has

Digitized by Google

been in the army, and is said to have had sunstroke in Egypt in 1882, but with this exception has had no other illnesses up to eighteen months ago, when he had something the matter with his kidneys. He denies having had syphilis. You will have noticed from his answers that there is a very marked alteration in his speech, with a tendency to slur over syllables, which sometimes makes it difficult to understand him. You will also notice from his replies that his memory is obviously deficient. (The patient failed for the time to remember important incidents in his life, but afterwards recollected some.) His wife tells us that his speech has been altering for some months, that his memory is bad, and that he tends to wander about aimlessly if he goes out by himself. He is said not to have any definite delusions. These mental symptoms, combined with the speech, suggest general paralysis, and on further examination the deep reflexes are found to be altered; in fact, the knee-jerks are quite absent on both sides. This illustrates the variations of the knee-jerks of which I spoke when we discussed the last case, where the reflexes were increased. The plantar reflexes are sluggish, but otherwise normal. There is a very slight tremor of the lips. Next we come to an examination of the eyes, and here, in this instance, we find no definite help, for the pupils are equal in size, and react, though slowly, to light.

These early cases of nervous disease are very important to study, since we often have an opportunity of seeing them and of making a diagnosis before the classical signs have all developed; and the earlier you have an opporunity of seeing cases the more certainly you will understand that the absence of any one symptom counts but little in any individual case. This point is particularly well illustrated in the numerous cases of locomotor ataxy attending from time to time, and in whom one sees many clinical variations in the beginning of the disease.

Comparing this case with the other, there are several points of interest. First there is the headache, which is important, because severe pains in the head are sometimes the most prominent early symptom. Then, again, this man is much more depressed than the other, and he has a sleepy, dull appearance, which is also a prominent feature in certain cases, the patients sometimes dropping off to sleep at all times of the day.

[Both these patients soon became worse and required to be placed under suitable control.]

A CLINICAL LECTURE
ON
ITCHING AS A SYMPTOM, AND ITS TREATMENT.*

By G. NORMAN MEACHEN, M.D., B.S.Lond., M.R.C.P.Lond.& Edin.,
Physician to the Skin Department, Tottenham Hospital
Assistant in the Dermatological Department, West London Hospital.

LADIES AND GENTLEMEN,—Itching of the skin is such a common symptom met with in everyday practice, and one which, next to pain, compels the sufferer therefrom to seek for relief as soon as it is felt, that I thought a brief consideration of some of the more important conditions in which it is present would be helpful. I shall also hope to show you illustrative cases from my out-patient department at this hospital.

In the first place, it is necessary to bear in mind that the causes of itching are very numerous and varied, so that when we are confronted by a case of pruritus we have to think of a great many things, and go through quite an elaborate mental process of exclusion until we arrive at what we consider to be the cause in the particular case before us. As a working classification for practical purposes, the accompanying table, adapted from Neisser, will be found, I think, the most useful :—

CAUSES OF ITCHING.

I. External	1. Mechanical irritation— (*a*) Parasites, (*b*) Contact with irritating substances. 2. Variations of temperature.
II. Internal	1. Toxic— (*a*) Hæmatogenous, (*b*) Gastro-intestinal, (*c*) Arterio-sclerosis, (*d*) Drugs. 2. Reflex, as in asthma, pregnancy, etc. 3. Nervous— (*a*) Neurotic, (*b*) In diseases of the nervous system.

* Delivered at the North-East London Post-Graduate College, Tottenham Hospital, N., May 21st, 1903.

Digitized by Google

III. Intra-cutaneous, *i. e.* in certain diseases of the skin accompanied by definite structural changes, as in eczema, psoriasis, lichen planus, etc.

Considered as a symptom, for it is no more a disease *per se* than is a cough or vomiting, itching is the subjective expression of irritation of the terminal nerve-filaments in the epithelium of the skin. The actual cause of the morbid sensation is believed by Bronson to be a disturbance of the sense of "pselaphesia" or contact. That this is the case in those varieties of pruritus due to external causes we can well understand.

The irritation felt may be so slight that it is hardly noticed by the patient at all, or it may be of such fierce intensity and persistence that the very existence of the unfortunate sufferer is rendered thereby well-nigh intolerable. Like most of the ills to which flesh is heir, pruritus, from whatever cause, is invariably worse at night, when the individual is warm in bed. From this it will readily be gathered that unrelieved itching of a week's duration is capable of making a patient look as haggard and as careworn as if he had been through a month's illness.

And here, in passing, we may observe one striking contrast between itching and pain; for whereas in the latter the instinctive tendency is to rest the affected part as much as possible, in the former there is an imperative desire for movement, in the shape of an immediate motor impulse to rub or scratch the irritating area. This may be an entirely unconscious act, as when we scratch our head when reflecting deeply; it may be a pure reflex, as in a young child suffering from scabies and whose inhibitory centres are as yet feebly developed; or it may be, and this is more usual, an intentional and deliberate conscious movement, designed for the express purpose of obtaining relief. And there is no doubt but that scratching does afford a real, and sometimes an almost pleasurable relief, temporarily, from all varieties of pruritus by helping to "work off" the accompanying surcharge of nervous energy. This brings us to what I may term literally

THE SIGN-MANUAL OF ITCHING,

namely, the *scratch-mark*. This, the commonest of all the secondary lesions of the skin, is a positive, visible, and undeniable proof of the presence of itching. It is generally a more or less linear streak, covered over by an adherent blood-crust, and tailing off to one extremity. It may also be accompanied by other lesions known as excoriations, which are more papular and irregular in shape, and which frequently go on to become pustular owing to microbic infection from dirty finger-nails or clothing. When you see these signs upon an infant's skin there is no need to ask the mother if she thinks the rash is irritable or not. You can see that it is for yourselves.

Here is a child suffering from that very common affection which has been variously styled "lichen urticatus" or "urticaria papulosa." She is eighteen months old, a very usual age for the disease to begin, showing that it is very closely connected with troubles of dentition and associated gastro-intestinal disorders. The mother will tell you that the rash starts like a "heat-bump" or a "water-blister," or "like a nettle-rash;" and if you look closely you will see that the initial lesion is a tiny papule or a small wheal, sometimes one and sometimes the other predominating. If a section of one of these early lesions be examined microscopically, we see that there is an acute dilatation of the papillary blood-vessels, with swelling of the papillæ themselves owing to the sudden distension of the smaller lymph-channels. The itching is in all probability due to the pressure thus exerted upon the minute nerve-endings in the deeper layers of the rete mucosum. The nerve-terminations may also be irritated by the presence in the blood-capillaries immediately surrounding them of some toxic material absorbed from the gastro-intestinal tract. The infant cannot restrain her scratching, and consequently we get the secondary lesions so plentifully exhibited in this child.

The treatment consists in removing any source of irritation from the alimentary canal, and in the local application of some soothing lotion. It is a good plan to begin by giving one grain of Hyd. c. Cret. with three grains of Sod. Bicarb. in form. pulv., half to be taken night and morning for two days. On the third day a mixture of two or three grains of salol, suspended in a little mucilage and flavoured with dill or anise, three times a day may be administered. The starchy elements in the diet must be reduced or even omitted altogether for a few days, all that is comprehended in the term

Digitized by Google

"cakes" being especially liable to undergo fermentative changes in the stomach and intestines. A little meat juice, broth, or albumen water may be substituted for these, often with advantage. Locally, an alkaline lotion containing five to ten minims of carbolic acid to the ounce will be found useful.

Before the time of Willan, the terms "pruritus" and "prurigo" were used indifferently to denote all affections of the skin accompanied by itching. In 1798 Willan first distinguished between pruritus, a simple itching without any cutaneous lesion, and prurigo, which is a papular disease accompanied by pruritus.

This little girl, aged four years, is a good example of *prurigo*. The disease is a definite clinical entity, somewhat resembling lichen urticatus, but occurring in rather older children. The mother states that the "spots on the legs" began six months ago, and by the numerous scratch-marks you will see that the itching has been great. The characteristic lesion is a small papule, which generally appears first upon the tibial surfaces, then upon the extensor surfaces of the thighs and trunk, the flexures of the joints being spared. Sometimes wheals are present. The glands in the groins are commonly enlarged, as in this case.

The treatment is very similar to that of papular urticaria, except that cod-liver oil and tonics are more frequently called for than in that affection. An ointment of two per cent. of β-napthol, a favourite application in Vienna, often succeeds well in allaying the itching.

There is another morbid state of the skin resulting from long-continued scratching, known as "lichenisation" or "lichenification," which was first described by Brocq. The skin becomes harsh, thickened, dry, and of a peculiar "stale," dirty brown colour, and is well seen in adults affected with pediculi corporis or with obstinate senile pruritus. Some of these patients may resemble, at first sight, those suffering from Addison's disease.

The most common external cause of pruritus is undoubtedly parasites; and, in fact, whenever we have to deal with a case of itching, the first step towards a correct diagnosis, and therefore scientific treatment, is to exclude, if possible, these unwelcome agents. Neither the social status nor the most cleanly habits of the patient should unduly influence us, for, unfortunately, none are proof against their invasion.

This woman is suffering from *scabies*, a disease very familiar to us all. It has a most ancient history, having been known for thousands of years. In the twelfth century the Arabians regarded it as being due to animals, called "syrones," under the skin. The earliest description of the parasite in this country was that of Moufet, published in 1634. As you will see by reference to this diagram, the acari live in galleries or tunnels in the horny layer of the epidermis, but very soon inflammatory changes of a more or less degree occur in the subjacent epithelium; hence scabies is recognised by Unna as a vesicular catarrh of the skin. The itching is due to the mechanical presence of the parasites, or, as some observers have considered, to a chemotactic influence exerted by them upon the blood-serum. The diagnostic feature is the burrow, or run, of which there are several to be seen upon this patient. It should be borne in mind, however, that in advanced and neglected cases they may not be visible owing to the presence of vesicles or pustules, and in such the acarus itself may fail to be discovered. A history of contagion may be wanting. It is in these instances that the secondary lesions are apt to mask the primary, and the case is in danger of being labelled as one of eczema or impetigo. The conditions may, of course, occur side by side as independent affections, when the diagnosis is rendered even more difficult.

When it is not desirable to give sulphur, either for fear of its increasing the dermatitis or on account of the social stigma associated with its use in the minds of some people, a most excellent substitute is the balsam of Peru. This can be employed in the form of an ointment in the strength of one or two drachms to the ounce, or it can be painted upon the affected parts directly. Some continental writers prefer it to sulphur as a routine method of treatment, believing it to be more efficacious.

Contact with various irritating substances may give rise to severe itching, which may precede the development of an urticarial eruption or an eczematous dermatitis. The "woolly bear" caterpillar so much prized by schoolboys, the *Primula obconica*, and the "poison-ivy" (*Rhus toxicodendron*) are examples of these exciting agents. That

Digitized by Google

variety of pruritus pudendi occurring in diabetics of both sexes, due to the irritation of saccharine urine, is also included in this group. I do not happen to have cases of this nature here to-day.

Variations of temperature and season are responsible for one troublesome form of itching known as *Pruritus hiemalis*, first described by Duhring in 1874. I am able to show you a case this afternoon in this man, a dairyman æt. 45. For the last six years he has suffered from an irritation of the skin, worse in cold weather and disappearing completely in the summer-time. There is no sugar in the urine, and his pulse-tension is not raised. If you examine his skin, you will notice that it is unusually dry, somewhat harsh, and the normal lines are intensified. In fact, he has a very mild degree of xeroderma and he has also had a slight attack of erythematous eczema upon his forearms. Upon questioning him, I found that he was wearing woollen clothing next his skin, and he had had recourse to one of the largely puffed varieties of soap which profess to "cure all skin diseases." I advised him to wear thin cotton next him, with a thicker material immediately over it, and to employ fine oatmeal in tepid water for purposes of ablution. At the same time he has been using an alkaline lotion of creolin, and an ointment of zinc oxide and lanoline for inunction. He will tell you that he has found great relief in so doing.

We will next consider some of the internal causes of itching.

Influence of Auto-intoxication.

Defects in the eliminatory apparatus of the body, and the consequent retention in the alimentary canal and general circulation of toxins, are responsible for many cases of pruritus which can be removed or greatly relieved by the administration of purgatives and diuretics. Besnier has long regarded it as an axiom that the majority of pruriginous affections of the skin are due to auto-intoxication, and the relationship has again been emphasised quite recently by Ehrmann, of Vienna. In a few cases of this nature increased quantities of indol and skatol have been found in the urine. It is a noteworthy fact, in this connection, that in a large number of cases of eczema the itching is invariably worse if the bowels are allowed to become constipated.

This patient, a married woman æt. 47, has suffered from relapsing *eczema* for two years. Just now the disease is almost quiescent. She will tell you that the itching is always more severe when the bowels do not act properly, and at the same time there is often an increased outbreak of the small papules, some of which you can still see upon the flexor surfaces of the arms. Magnesium sulphate is, I think, the best drug to give under such circumstances, in drachm doses every two hours, until the bowels are moved. A tumblerful of one of the many excellent aperient waters on the market sipped slowly while dressing in the morning, may be all that is needed to ensure the regular elimination of waste products.

As regards local treatment, the first place must be given to carbolic acid, which rarely fails altogether to give relief in pruritus, from whatever cause. A drachm to the pint of water forms a useful lotion which may be dabbed on the affected parts frequently. If erythema be present as well, the pruritic area may be covered by plain sterilised gauze or lint soaked in the following cream or emulsion:— ℞ Zinci Oxide, 3 dr.; Acidi Carbolici, 20 m; Mucilag. Acaciæ, 2 dr.; Emuls. Amygdalæ, 2 oz.; Aquam, ad 8 oz. At other times menthol or thymol will be found more acceptable. They may be used in the form of lotions or ointments in the strength of two grains to the ounce.

The question of soap is a vexed one, and a full consideration of the many different varieties which have from time to time been "recommended by the Faculty" would lead us too far from our subject. Suffice it to say, therefore, that, as a general rule, any soap is harmful in pruriginous eczema accompanied by papules, vesicles, or much weeping. The soothing and at the same time cleansing properties of bran or fine oatmeal will be found of greater benefit in such cases. Where there is simply itching in a harsh, dry skin, in the condition which may be termed "pre-eczematous," a super-fatted soap of menthol or cold cream with oatmeal may be employed with advantage.

The next patient, also a married woman, æt. 55, exhibits another form of pruritus, namely, that associated with high arterial tension. She has been troubled for three years with a general

Digitized by Google

itching of the skin, dizziness on rising in the morning, and "rheumatic pains" in the limbs. She is said to have had "gout" twenty years ago. There is little to be seen upon the surface of the skin except a few scratch-marks, and parasitic causes can be excluded. You will notice that the forearms and neck are slightly erythematous and rough, a soil favourable for the development of eczema of the "washerwoman's" type. If you feel her pulse, you will agree with me that its tension is increased. The urine has a specific gravity of 1020, and contains a trace of albumen.

As an early sign of granular kidney, pruritus may be seen in common with polyuria, cramps in the legs, headaches, and epistaxis. It is supposed that the itching is due to the irritation of the nerve-filaments by toxins circulating in the neighbouring capillaries, and which have failed to be excreted owing to the renal insufficiency. The success of treatment which aims at restoring the impaired excretory processes in relieving the pruritus would seem to support such a theory.

The intolerable itching seen in jaundice is sometimes very difficult to control. Lotions of carbolic acid give relief for a time, as also may lead and opium. Thyroid extract internally administered has been recommended of late on theoretical grounds, but I have had no experience of it as a remedy in this connection.

There are many *drugs* capable of producing itching of the skin. The most common are opium, alcohol, cocaine, coffee, and arsenic, in order of frequency. Some of the cases are only clinical curiosities, while many remain undiagnosed, the condition being ascribed to other causes.

Cases of *reflex pruritus* are not numerous, though it is important to recognise them. A boy of six came to the out-patient department with itching of the skin which he had had for six weeks. All the usual causes could be eliminated, but from his mouth-breathing and high-arched palate I suspected that he probably had adenoids. These were kindly removed by my colleague, Mr. Carson, and from that time the mother stated that the irritation ceased. I could only conclude that this was one of those cases of pure reflex itching which disappears with the removal of the focus of irritation.

The condition known as *prurigo gestationis* undoubtedly comes under this category, though in some cases the question of auto-intoxication has also to be considered. Young women in the first month of pregnancy are chiefly affected, and sometimes the disease recurs with increasing intensity in each successive pregnancy. In a patient æt. 22, sent to me by Dr. Spears, of Wood Green, the itching started upon the legs and lower part of the trunk at about the tenth week, and it had proved very rebellious to treatment. Free regular action of the bowels must be ensured in these cases, and alkaline lotions of creolin, in the strength of one to two per cent., will be found useful for local application.

When we take into consideration the importance of the mental factor in influencing or modifying physical processes and sensations, it is not surprising to find that the feeling of itching may be profoundly affected by certain mental conditions. The late Sir Erasmus Wilson drew attention to the marked neurotic character of some patients troubled with severe pruritus; and he used to relate the story of a respectable widow whose sufferings were increased tenfold by the supposition of some of her medical attendants that her condition was due to pediculi! I had hoped to show you a case today in which the idea of being invaded with parasites had so firmly taken possession of the patient that it almost amounted to a delusion. This condition, which is known as "phtheireophobia," analogous to syphilophobia, is more common in women who have just reached the menopause. It may have started as an actual pediculosis, but, although the cause be removed, the sensation of itching persists, or is imagined to persist, until the idea may become fixed in the mind. Such cases may even drift into asylums. The treatment in the early stages is chiefly moral, active suggestion being brought to bear upon the patient that the causes of her trouble no longer exist. The general nerve tone must also be attended to; bromides may be given to quiet nervous excitement, while plenty of rest and nutritous diet should be secured.

Pruritus may be met with as one of the protean manifestations of functional nerve disorder, as in neurasthenia. It is also seen in some organic nervous diseases, especially in locomotor ataxy, though more commonly in its exaggerated and modified form of tingling or formication.

Digitized by Google

Regarded as

A Symptom of Cutaneous Disorder,

pruritus is very frequent, and is one for which patients early seek advice. How often do we not see a more or less violent itching usher in an attack of eczema? And again, when the same disease is fully established, paroxysms of itching and smarting are prone to disturb the sufferer's rest, and are sometimes most difficult to overcome. Look also at the severe pruritus encountered in certain cases of psoriasis, in urticaria, in pemphigus, and even occasionally in ulcer of the leg. One of the first questions we ask of a patient with any skin disease is, "Does it itch?" Yet there are many eruptions in which this distressing symptom is not complained of, or only seldom.

The presence or absence of itching is explained, to a large extent, as Bronson has pointed out, by the morbid anatomical features of the disease. Thus it is found that those disorders which chiefly affect the corium, and do not appreciably disturb the prickle-cell layer of the epidermis, very seldom itch. Of such are lupus and syphilis, in both of which it is rare to find marked pruritus, unless secondary changes involving the epithelium have supervened. On the other hand, practically all papular, vesicular, pustular, and bullous diseases which have their origin in the epidermic layers are characterised by itching during some part of their evolution.

It may be asked why itching is not more common in herpes zoster, which is surely a vesicular disorder. All that can be said in answer to that is that as herpes is definitely related to organic changes in the nervous sytem, the sensation of itching which we might have expected is transformed into one of pain, *i.e.* into burning and smarting, which therefore predominates.

Next to eczema, the skin disease in which pruritus is apt to be a troublesome symptom is *seborrhœa*, in one or other of its forms. It is the most common cause of itching of the scalp, particularly in adults. Clinically, you are familiar with the fawn-coloured, greasy, slightly scaly patches in the sternal and interscapular regions, formerly known by the not altogether inappropriate name of "flannel rash." The constant wear of a non-porous flannel material next the skin, especially if seldom changed, is undoubtedly responsible for such eruptions in individuals who sweat much. But, as the majority of the cases show distinct evidence of seborrhœa in the scalp, and as many of them also suffer from facial acne, there are strong grounds for believing that the process is an infective one spreading from above. This theory is confirmed by the discovery of Unna's micro-bacilli in the lesions.

This woman is married, æt. 28. For the last seven years she has suffered off and on from great irritation of the skin accompanied by a "rough rash" all over. When I saw her about six months ago, there were numerous discrete spots of seborrhœic dermatitis on the trunk and limbs. They did not show any marked preference for the extensor surfaces, and they were not scaly enough nor of the right tint for psoriasis. Moreover the scalp was very scurfy. Her trouble has improved greatly under the use of creolin baths and an ointment of twenty grains each of salicylic acid and sulphur to the ounce of vaseline. Her scalp is now practically clear.

When the head is much affected, a preliminary shampoo with the spiritus saponis kalinus, repeated on successive days if necessary, is useful in removing scales, after which an ointment such as the above can be rubbed in. The addition of five to ten grains of ammoniated mercury will often render it more efficacious. In other cases resorcin appears to act better, either as a five per cent. alcoholic lotion or in ointment form.

The itching which frequently accompanies *lichen planus* is sometimes very severe, and, as you will see under the microscope, there are definite pathological changes to account for it, among which the flattening of the papillæ and the hyaline condition of the epithelium are the most prominent.

This patient, a single woman æt. 36, had lichen planus a year ago, and two months ago she came to the hospital complaining of itching, confined to the legs, where there are still numerous areas of pigmentation visible resulting from her former trouble. Sometimes an attack of pruritus heralds the onset of lichen planus, but she has not, fortunately, had any fresh outbreak. She has been using a lotion of carbolic acid and glycerine.

A special form of itching occurring in elderly subjects has long been differentiated as *pruritus senilis*. This term should, strictly speaking, be reserved only for those idiopathic cases where no

Digitized by Google

cause can be ascertained to account for the itching, as there are many cases which are called senile pruritus that are in reality due to pediculosis or eczema. It is the natural outcome of senile degenerative changes in the skin. These consist mainly of a granular degeneration of the collagenous or connective-tissue elements, together with a thinning of the epidermis.

This old lady, æt. 71, has been troubled with irritation of the skin for three months. There are no visible cutaneous lesions, and there is no history of alcoholism. The pulse-tension is not markedly increased, and there is some senile tremor. She has found great benefit from the outward application of an ointment of ten grains each of menthol and zinc oxide to the ounce of lanoline.

The treatment of this troublesome variety sometimes taxes our therapeutic resources to the utmost. The patient often gets into a state of great nervous irritability through insomnia, and we have this to contend with as well.

Internal medication is of great value, and much can be done towards promoting a cure by securing sleep and composing nervous restlessness. For this purpose, small doses of cannabis indica are most useful. The coal-tar derivatives, such as antipyrin, are not to be recommended, not only on account of their depressing action, but because they themselves are liable to produce cutaneous eruptions in susceptible individuals. Many cases are greatly benefited by mild purging, followed by the administration of intestinal antiseptics, such as salophen or β-napthol. I need hardly say that any abuse with regard to coffee or alcohol should be checked.

Locally, the "substitution treatment," as it is termed by Bronson, is the most satisfactory and rational. It consists in distracting the attention, so to speak, of the cutaneous nerves by the substitution of another sensation for that of itching. Thus the sensation of cold or numbness is produced by carbolic acid, menthol, or by the evaporation of spirituous lotions. On the contrary, the soothing effect of heat in the relief of itching is one which is often taken advantage of by patients themselves. It is true that the beneficial result derived from this source is only temporary, but five minutes' immersion in water as hot as can be borne comfortably, just before getting into bed, followed by rapid drying and sprinkling of the parts most affected with a dusting powder of starch and zinc oxide, or dermatol, may make all the difference between a good and a bad night. In other cases, mechanical friction of the skin with a soft brush, as suggested by Jaenicke, has proved of service. The dried-up epithelial scales so often seen upon the harsh skin of senile pruritics is thus removed, when a little lanoline can then be gently rubbed in.

If we come to think of it, all antipruritic applications that are really efficacious act upon this principle of "substitution," and yet we could hardly apply the term "rational" to the mode of action of every one. The treatment of itching is, and must ever be, largely symptomatic, and therefore some more or less empirical remedies still, in our present state of knowledge, find their place in our armament; but they need be none the worse for that.

It is only when all the above causes of itching can be excluded in any given case that we are justified in calling it idiopathic, and this word, like many others in medicine, is fast disappearing from our vocabulary.

There are many other points connected with this subject which one would like to mention, but as time will not permit, I must reserve them for a future occasion.

We have received from Messrs. Burroughs Wellcome & Co. a specimen of "Tabloid" Antifebrin Compound. The formula for this preparation is as follows:

Antifebrin (Acetanilide) . .	gr. 2.
Caffeine Citrate	gr. 1.
Camphor Monobromide . .	gr. 1.

This combination has been found useful in various forms of headache, in neuralgia, acute rheumatism, and other painful affections. It has also been prescribed with advantage in obstinate cases of vomiting following operations, and in sea-sickness.

"Tabloid" Antifebrin Compound may be prescribed in any of the cases in which acetanilide is indicated.

The advantage of this preparation is clearly indicated from its formula, and it will be found that it will be largely prescribed for the purpose of procuring its antipyretic and analgesic effects.

Digitized by Google

SQUINT: ITS CAUSES, PATHOLOGY, AND TREATMENT. By CLAUD WORTH, F.R.C.S. (London: John Bale, Sons, and Danielsson, Ltd., 1903.)

Mr. Claud Worth is to be congratulated upon the excellence of his valuable monograph on squint. In it there is throughout abundant evidence of most careful clinical observation; the facts and conclusions set forth are the outcome of the examination of over 2300 cases of squint and heterophoria, extending over a period of ten years' work at some four London hospitals. The detailed account of the method of training the "fusion sense" by the instrument known as the amblyoscope, devised by the author, is worthy of careful study. In Chapter IX is a complete account of the progress made in the treatment of sixteen cases of squint where the non-seeing eye is trained to take its share in vision.

The author's operation for "advancement" is very clearly described, and is one which ophthalmic surgeons will do well to perform; the figure representing the stages in the operation, on page 208, is a very useful one, and shows the position of the parts from the operator's point of view. The book is worthy of the attention of all surgeons interested in the treatment of squint.

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY.—A new and complete dictionary of the terms used in medicine, surgery, dentistry, pharmacy, chemistry, and the kindred branches, with their pronunciation, derivation, and definition, including much collateral information of an encyclopædic character. By W. A. NEWMAN DORLAND, A.M., M.D. Together with new and elaborate tables of arteries, muscles, nerves, veins, etc.; of bacilli, bacteria, diplococci, micrococci, streptococci, ptomains and leukomains, weights and measures; eponymic tables of diseases, operations, signs and symptoms, stains, tests, methods of treatment, etc. With numerous illustrations and twenty-four coloured plates. (W. B. Saunders & Company, London and Philadelphia.)

We have given the full title-page of this excellent dictionary because it admirably indicates the nature of the contents of the volume. The book is well bound and clearly printed, whilst the illustrations and the twenty-four coloured plates set an example of finished work that is worthy of much commendation. It is of interest to note at the present time that Dr. Dorland describes "tabloid" as "a proprietary word." A considerable amount of trouble and labour must have been expended in the arrangement and production of the list of bacilli; and the dictionary has every quality entitling the author to describe it as complete, accurate, and up to date.

THE PRACTICE OF OBSTETRICS; designed for the use of students and practitioners of medicine. By J. CLIFTON EDGAR, Professor of Obstetrics and Clinical Midwifery in the Cornell University Medical College. With 1221 illustrations. (Rebman, Limited, 129, Shaftesbury Avenue, London.)

The aim of this book is to present the subject of midwifery from a practical and clinical standpoint, so that it will best facilitate the requirements of the student of medicine and of the active obstetrician. Professor Edgar has in our opinion very wisely adopted the simplest classification. He has also omitted as unnecessary for the purpose of this work the usual elaborate section upon the anatomy of the female genital organs, and has directed his attention to the physiology of these organs. The author points out in his preface that one object of the omission of this separate section on anatomy is to avoid repetition, for, as he very truly observes, the histological and topographical anatomical details required will be found in their appropriate places under the headings dealing with physiology, pregnancy, and labour. In order to adequately comment on a book containing more than 1100 pages more space is required than it is possible to give, but the extremely practical and accurate nature of the work can be judged from the fact that the illustrations of vulval engagement of the presenting parts in physiological pregnancy are from flash-light photographs. A valuable portion of the book consists of short sections upon the medico-legal aspects of obstetrics, and Professor Edgar has something of importance to say on the difficult subject of the determination of sex. The large experience of the author will ensure considerable attention being given to his opinion that "radiography in obstetrical practice is still in its infancy, and the results as to fœtography have been disappointing. On the other hand, Röntgen photography of the maternal pelvis is a highly promising field, but as yet offers no practical advantages." Care has been taken to avoid as far as possible full-page illustrations, and to insert the illustrations in the midst of the text itself, so as to more readily catch the reader's eye. We suggest that in the next edition of the book a list of the 1221 illustrations should be included.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 581. WEDNESDAY, DECEMBER 16, 1903. Vol. XXIII. No. 9.

CONTENTS.

PAGE

*A Lecture on Albuminuria. By P. H. PYE-SMITH, M.D., F.R.C.P., F.R.S. ... 129

*Three Clinical Lectures on Head Injuries. By T. P. LEGG, F.R.C.S. LECTURE I.—Injuries of the Scalp and Face. 137

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

A LECTURE

ON

ALBUMINURIA.*

By P. H. PYE-SMITH, M.D., F.R.C.P., F.R.S.,
Consulting Physician to Guy's Hospital.

GENTLEMEN,—I propose to-day to say a few words upon albuminuria, and will mention at once the limits within which I ask your attention. The pathology of albuminuria is a most interesting and important subject, but bearing in mind the limits of time, I do not propose to enter upon it now. What I wish to say a few words upon are, first, the different proteids which are excreted by the urine —those which are practically important, and those which are not; the methods of detecting them, the most useful clinical tests for albuminuria, and then the diagnosis as to its seat and significance. Lastly, I propose to consider the prognosis of albuminuria, with especial reference to the more difficult cases which come before us as examiners for Life Assurance.

When one used to ask students who discovered the frequent presence of albumen in the urine, the most intelligent used to say Dr. Bright. The history of discoveries is not only interesting, but instructive. Towards the end of the eighteenth century albuminuria was recognised by Cruikshank, and its association with dropsy was known to Wells of St. Thomas's, to Osborne of Dublin, and to Dr. Blackall of Exeter, who died at an advanced age in 1840.† He was aware that in certain cases of dropsy the urine when heated would become opaque, and coagulate—the same reaction as takes place every day when an egg is boiled. It was known that the serum of the blood

* Delivered at the Medical Graduates' College and Polyclinic, March 2nd, 1903.

† 'Observations of the Nature and Cure of Dropsies, and particularly on the presence of the coagulable part of the blood in dropsical urine,' 1813.

Digitized by Google

when heated would coagulate, and it was very naturally supposed—and I do not know whether even now we can say it was an incorrect way of putting it—that in certain kinds of dropsy serum appeared in the urine. Blackall's explanation was that it was an effort of Nature to relieve the disease by taking up the effused fluid—the serum of ascites and anasarca—and getting rid of it through the kidneys. This also was a natural supposition, but it had two faults. First, it assumed that unfortunate hypothesis of an effort of Nature to antagonise disease, which even now is still brought forward as explaining pathological processes. Surely we have got far beyond that. We no longer believe that Nature (written with a large N) is a benevolent female with large breasts and benign countenance, who makes well-intentioned but often ill-directed efforts against disease. Surely that is an utterly unscientific conception. There is no such thing as Nature in this sense, and an effort of Nature, *i.e.* a reaction to a disturbing cause, is as often hurtful as beneficial. Besides, it is as "natural" to die as to be born, and the same all-wise Providence that gives us health gives us also sickness. What we find is that with dropsy, as with paralysis or dyspnœa, certain natural processes follow injury, or poison, or parasitical invasion; and while these effects are sometimes beneficial, they are sometimes just the reverse. In either case they are the direct reactions to a physical, or physiological, or chemical disturbance. They have no final purpose, and can have none; and therefore to depend upon the *vis medicatrix naturæ* is to trust to a figment of bygone time. This was one fault of the theories that albuminuria was due to reabsorption of the affected serum by an effort of Nature to get rid of the dropsy.

The other fault was that it was a mere explanation without any attempt to verify its accuracy. Now, as the late Sir William Gull used to say, "savages explain, scientific men investigate." It was Bright who found the real significance of albumen in the urine, and that he did by the very ordinary method of not framing hypotheses but collecting facts. Every patient who died of dropsy, and in whose urine albumen was found, he followed to the deadhouse. He looked at the heart, and he looked at the kidneys, and based upon those observations the definite conclusion that there was a constant association between coagulable urine, dropsy, and obvious anatomical changes in the kidneys after death.

Another historical point, if you will forgive me for dwelling upon these matters, I had from the late Dr. Owen Rees, who with Dr. George Barlow was assistant physician at the same hospital, and who aided Dr. Bright in his researches. The whole discovery was very nearly being shaken by the fact that in many cases, though the urine became opaque on boiling, they found the kidneys healthy after death. Bright was on the point of abandoning the investigation as not leading to constant results; but Owen Rees was a good chemist, and knew that earthy phosphates would come down when a solution was heated, and also that these phosphates were soluble in acid. In some of these cases when it was found that the kidneys were not diseased he went back to the specimen of the urine, and showed that phosphates and not albumen caused the opacity on heating. He thus came upon the fallacy of mistaking earthy phosphates for albumen, and preserved Dr. Bright from what at the onset of his observations would have probably spoilt his work.

Our next question will be, do we mean anything more by the word albuminuria than Bright meant? We know that albumen is not exactly the same as white of egg. There are differences of diffusibility and solubility, which separate serum-albumen from ov-albumen. We also know that beside serum-albumen there is serum-globulin present in many cases of Bright's disease, though I do not know how many.

The separation of albumen from globulin in cases of albuminuria by a saturated solution of magnesium sulphate is a tedious process and of little practical interest. In a large number of cases, probably in half, the precipitate we get with heat is a combined precipitate of serum-albumen with more or less serum-globulin. But there does not appear to be any important diagnostic help gained from the proportion of globulin present.

There are beside globulin two or three other proteids which may be present in urine, but they also are not of great practical importance. In the first place, we have those very soluble and diffusible proteids which used to be called peptones, but which now are more accurately called albumoses, true peptones being extremely rare in the urine. The albumoses, which mark a step towards the formation of peptones, are found more often, but not in cases

Digitized by Google

of Bright's disease. They occur in pyrexia, as in enteric fever and pneumonia, and in some forms of multiple tumours, and sometimes during digestion in healthy persons. A common fallacy in testing for albumen is the presence of what used to be called mucin, from the genito-urinary tract. Mucin is a mixture; the proteid present in mucus, and also in pus and other conditions, is now known as nucleo-albumen or nucleo-proteid. It frequently is present with a trace of albumen, and sometimes without any albumen at all. Nucleo-albumen is held in solution by salines, and is rendered insoluble by addition of distilled water or by addition of acetic or citric or picric acid, and is redissolved by alkalies. It is almost always evidence of irritation of the bladder, urethra, renal pelvis, or vagina, and it probably never points to disease of the kidney itself.

Now we come to the test for "albumen," *i. e.* for the mixture of serum-albumen and globulin which is present in Bright's disease. I believe that the oldest test is also on the whole the most valuable and the least liable to fallacies, namely, that of heat. It is certainly the most delicate. For many years I used to try every case of albuminuria by four tests, and became convinced that the test by boiling is the most delicate and trustworthy of all. Next or almost equal in delicacy comes the test by picric acid; thirdly, that by nascent ferrocyanic acid; and last and least delicate, that by nitric acid. This does not, however, by any means decide which is the best test, because we do not want in most cases to detect the smallest trace of albumen, which might be of little pathological importance. So we want something which will be certain as well as delicate.

What are the fallacies with regard to testing by heat? The first we are familiar with, the fallacy of the phosphates coming down; the opacity is at once distinguished by adding a drop of acetic acid. If the urine is alkaline it should be first neutralised, or a small amount of albumen may escape detection; but if too much acid is added, acid-albumen will be formed, which is not precipitated by heat.

The next most commonly used test is that of nitric acid. By far the best plan is to put the nitric acid first into a rather long narrow test-tube, and slowly run the urine down upon it, and then the characteristic white ring forms at the junction of the heavier and lighter fluid. The chief disadvantage is trivial but important. It is that you cannot conveniently carry nitric acid about; it is too corrosive. It is only fit to be used in one's room, and even there you must be careful not to stain your fingers or clothes with it. Another point is that the nitric acid takes a little time to act, and sometimes it will be necessary for you to wait for five minutes before the ring is well marked. Then the patient may have been taking cubebs or copaiba, and that causes a precipitate with nitric acid. This fallacy is easily recognised by the heat test. I remember a case in which a young man came to the hospital with a rash which was regarded as scarlet fever, and albumen was found in his urine. The clinical clerk had tested with nitric acid, and it turned out afterwards that the urine contained not albumen but copaiba, and that the supposed scarlet fever was really a secondary rash.

With regard to the acetic acid and ferrocyanide of potassium test, that is a very convenient one, and has scarcely any fallacies. It is a test which can be carried about easily by the help of Dr. George Oliver's slips of paper or Dr. Pavy's pellets. You need not even take acetic acid with you, but only ferro-cyanide; for there is vinegar in every house, which will do as well as the acetic acid of the Pharmacopœia.

We now come to reaction with picric acid. This test is popular and has good qualities; it acts more quickly than nitric acid, and there is no difficulty in carrying it about. It is quite delicate enough for most purposes, almost as delicate as that by heat. The objection to it is that it does not definitely prove that albumen is present. On adding picric acid with strong acetic acid to urine in a tall narrow tube, a certain opacity follows if nucleo-albumen is present, or there may be a precipitate of urates, or occasionally of uric acid. Another fallacy, I am sure, has often led to mistakes. If you add a saturated solution of picric acid to alkaline urine you do not get a precipitate, although albumen is present. I have tried it with amphichroic urine (a not uncommon condition) containing an appreciable quantity of albumen, and this test has failed to show a cloud even after ten minutes. So you must always add a vegetable acid, such as citric acid or acetic acid.

Lastly, you may see a granular yellow precipitate of picric acid gradually form and sink to the bottom

Digitized by Google

of the test-tube. It is obviously not albumen, and dissolves in potash.

But if you do not get a reaction with well-acidulated picric acid, you may be sure that the urine is healthy. In out-patient practice it is convenient to have some test which will readily exclude the presence of albumen. In assurance work, also, it is very convenient to have a test which you can apply, and say at once that the urine of the applicant is free from albumen. You cannot be sure that albumen is present if you have a precipitate, but must test it by other methods.

Many other tests for albumen have been proposed One was introduced by the late Sir William Roberts, consisting of a strong brine into which the urine was dropped. There was one by Dr. Rees, namely, tincture of galls, which is a delicate test and easy to carry out. Then there is salicyl-sulphonic acid, which at one time was much used, and tungstate of sodium.

As to the amount of albumen in the urine, it is laborious, and I think unnecessary, even in the laboratory, to collect and weigh the precipitated albumen. The weight is very small, even where the urine seems to be solid with albumen on heating. Most of us have used Esbach's graded test-tubes, by which after testing we can measure off and ascertain the amount of albumen. The objection is that the bulk of the precipitated albumen varies according to the time. You must leave it for twenty-four hours; and if, after you have measured it, you leave it for another twelve hours, you find it has shrunk, so as to give a different reading. And after all I doubt whether there is much gained by arriving at what is really only an approximation to the quantity passed. It is not the quantity of albumen passed which is of importance, it is the fact that albumen is constantly present in the urine. Moreover, if we are to make a useful estimation of the urea, or the albumen, or the sugar, or any other constituent in the urine, the whole twenty-four hours' urine must be collected. That is a very proper thing to do in hospital wards, but, as you know, it is almost impracticable in private practice; and we want to make examination of the urine as easy for our patients as possible, and not as difficult.

We now come to consider the conditions of which albuminuria is an indication. Some say that there is such a thing as physiological albuminuria. It may be a question of words, but I would never use that term at all. Physiological means that which occurs in perfect health. No doubt it is impossible to draw an exact line between health and sickness; they overlap one another. I suppose none of us in this room can say we are absolutely healthy; we are not as healthy as we were when we were children; but still the difference between the state of health in which the urine does not contain albumen, and the state in which albumen is present is a very important one. No one has albumen in his urine without having something wrong about him, which is quite worth the attention of his medical adviser. It is therefore dangerous to speak of the presence of albuminuria as ever being a physiological condition. To put it in another way, no healthy person can produce albuminuria by anything he can do,—by moderate exercise, by eating eggs, or by any other means. It is always a morbid symptom.

The next point to determine is, at what point the albumen has entered. Was it in the urethra, or in the bladder, or in the pelvis of the kidney, or in the secreting substance of the gland? The larger number of cases called physiological albuminuria in young men depend, I believe, upon the presence of nucleo-albumen from mucus or pus or semen in the prostate, or the bladder, or the urethra. In the case of women a similar condition is most often due to mucus or pus in the vagina. When you find "a trace of albumen" in a person otherwise healthy, and with no changes in the pulse or in the heart, you may probably ascribe it to such cause. The microscope is of help in instances of that kind. If you do not find casts, but do find pus corpuscles, you are very much confirmed in the view that the kidneys are unaffected. There are few conditions in which we have learnt more of late years than from the staining of the tubercle bacilli in cases of cystitis. When one first learnt that the tubercle bacillus was to be found in the sputum, one was disappointed again and again in not finding it in the urine, even in cases of clearly tuberculous cystitis or pyelitis. But the application of the centrifuge for this purpose has been most successful, and now it is not the exception but the rule to recognise the presence of tubercle bacilli in those cases.

Next we come to toxic albuminuria, which we must always be on our guard against forgetting.

Digitized by Google

Many drugs will produce it; perhaps turpentine and cantharides are the most striking and the most rapid in their effects. Very often they produce hæmaturia also, but not always.

Then there is the febrile form of albuminuria, which is very important, because it is desirable for the purposes of prognosis that we should separate it carefully from the albuminuria due to disease of the kidneys. As we all know, there is no form of pyrexia which may not be accompanied with more or less of albumen. In my own experience, when many years ago I was registrar of a large hospital and tested the urine of every patient who had a raised temperature, I found albumen most constantly, almost universally present in cases of erysipelas. I found it least constantly present in chronic phthisis. Between those two diseases I put pneumonia next to erysipelas. It is very seldom you will not find albuminuria in acute pneumonia if you look for it. I do not refer now to broncho-pneumonia or tuberculous or chronic fibrous disease, but acute lobar pneumonia. That disease I believe is almost always accompanied by the presence of albumen, and there is sometimes a large amount present. In enteric fever, again, the more carefully one looks for it the more often it will be found, and my impression is that there are few cases of enteric fever in which a careful daily observer will not find albumen present. In typhus, when one saw more of it, albumen was, I believe, always present. In diphtheria, too, albuminuria is constanty present, except in the mildest cases. We recognise albuminuria as due to the febrile condition, and not to any change in the kidneys, first by its being moderate in amount; secondly, by it being unaccompanied by a hard pulse, or by any sign of defect in the heart; thirdly, by there being no casts present; and lastly, by its passing away as the patient recovers. The only real difficulty, it appears to me, is in cases of scarlet fever. No doubt you may find a considerable amount of albumen, as much as in diphtheria and pneumonia, where there is no reason to suppose that any permanent mischief will befall the kidneys. The child recovers and the albumen disappears, just as it would after typhoid fever. This is a febrile albuminuria. But during convalescence we unfortunately often see an acute nephritis following with a different kind of albuminuria, that with casts, and very often with blood, while it lasts for a long time, and is associated with definite anatomical changes in the kidneys. That is almost the only case in which, as far as I know, febrile albuminuria is followed by a permanent organic disease of the kidney. In diphtheria the same thing very occasionally happens. I have had two cases in which I suspected this cause of Bright's disease. I believe that pathologically these two conditions are quite distinct—the slight albuminuria during the progress of the fever, from the subsequent albuminuria due to nephritis. If I am right—and I feel confident of it, having watched cases from that point of view—one need not be alarmed by albuminuria occurring in febrile diseases, and one may confidently expect that if we get the patient safely through the disease the albumen will disappear from the urine as the temperature goes down.

Then we have those familiar cases in which albuminuria is due to excessive congestion of the kidney, as in mitral incompetence, or in chronic emphysema with dilatation of the right side of the heart. In cases of cardiac disease with dropsy and albuminuria it is sometimes hard to say whether the kidneys are diseased or not. The rules are sufficiently plain; where the urine is scanty and high-coloured, and the amount of albumen considerable, with perhaps a little blood in the urine, with few hyaline casts or none at all, then we are entitled to believe that that is only congestion of the kidney, with no structural changes, and we venture to use mild saline diuretics, with perhaps some blue pill. The patient recovers, and passes urine which is perfectly healthy. But if we find the urine abundant, with epithelial or fatty casts; if we find that the pigment is small in quantity—that is to say that the kidneys are not doing their work in excreting,—and if during the progress of the disease the cardiac symptoms improve but the albuminuria remains, then we come reluctantly to the conclusion that the heart disease is complicated by a separate and independent affection of the kidneys, which very much limits our powers to treat it, and makes the case far more grave. In some cases, however, we must ask whether a patient who has cardiac symptoms with albuminuria is not suffering from primary Bright's disease, with secondary dilatation of the left ventricle. We know the abundant pale urine, little more than water, which comes from a patient with contracted kidneys; we

Digitized by Google

know that it is associated with a hard pulse, hypertrophied left ventricle, and tendency to hæmorrhages. Perhaps it is not so well known that in such cases blood may appear in the urine. No doubt blood often appears in the urine when there is a "large red kidney," as in the "active" congestion of scarlatinal nephritis; but sometimes it is due to atheromatous renal arteries, which bleed, just as the patients with contracted kidneys are liable to hæmorrhage in the brain, and small hæmorrhages in various parts of the body.

Now we come to the mildest kind of albuminuria, the kind which is sometimes called physiological, and which was termed by Dr. Pavy cyclical albuminuria. This was first described by the late Dr. Moxon as the albuminuria of adolescence, and it has been the subject of many investigations in this country, in Germany, and in France, among soldiers, sailors, policemen, schoolboys, and boys on training-ships. It almost always occurs in males, usually in boys and young men. If you read the accounts of these cases critically you will suspect that some of them are not true renal albuminuria. For instance, the late Sir Grainger Stewart in Edinburgh made observations upon 200 Scottish soldiers in garrison in Edinburgh Castle, and he found a large number had traces of albumen in their urine. They were strong, healthy men, without the least evidence of Bright's disease, and I cannot help believing that a little scepticism and careful investigation would have shown that most of those cases were of urethral or prostatic origin, and not cases of renal disease. So also many of the cases which come before one in insurance work, and which occur in young men and youths, are due to contamination of the urine, either with pus or with mucus, or with spermatozoa. For these causes you must make considerable allowance, as Dr. Moxon himself admitted. But there remain many cases in which thorough examination shows there was no mucus, no pus, no blood, and no spermatozoa. These cases, as far as I have seen them, are most common in boys from twelve or fourteen years of age up to the time they leave school at eighteen or nineteen. Some of them are clearly the result of the application of cold to the surface of the body, as from cold bathing, or after bathing the boys have got into the water again, until at last they are chilled through, with blue fingers and noses, and trembling while they dress. One proof is that occasionally those boys pass with the albumen a little colouring matter,—that is to say, methæmoglobin. I have had two patients, boys from different public schools, between the ages of sixteen and eighteen, both liable to occasional attacks of hæmoglobinuria. These boys after bathing often passed urine with a little albumen in it; but at other times there was no albumen present, and the boys were otherwise in good health. Sometimes I have known driving in the cold air produce temporary albuminuria.

Another cause is blowing wind instruments, which no doubt may cause considerable venous congestion in the kidneys as well as elsewhere. That has been observed in regimental bands, and in brass bands connected with Boys' Homes and training-ships. Again I have seen, once or twice, undoubted albuminuria of a transitory character, along with high-coloured urine and excess of lithates, after unusual exertion, such as after playing football, or cross-country running. In another case, a man who used to enjoy hunting in Leicestershire, instead of coming home the better for his holiday, was worse. He was found to be passing some albumen, and with scanty high-coloured urine and abundance of uric acid. Eventually this congestion of the kidneys passed away without leaving any trace of renal disease.

Another form of so-called "physiological" albuminuria appears while dressing in the morning. This may be due to getting out of a warm bed and stripping to wash in cold water, and then, perhaps, delay in putting on clothes. This, in a susceptible person, may cause congestion of the kidneys, and albuminuria in consequence.

We now come to the question of prognosis, especially, as I said in my opening remarks, with regard to insurance. In early days the urine was only examined when dropsy, or other signs of Bright's disease, were present. Nowadays, I suppose, we all examine the urine before we prescribe or commit ourselves to a diagnosis. All insurance offices in this country and in America now insist upon the urine being examined in every case, and that, no doubt, is the only proper thing to do. But those who have any experience of this branch of practice will agree with me, that there are few cases more difficult than for a man, apparently in good health, robust and vigorous, to be rejected on

Digitized by Google

account of a little albumen in the urine. Or sometimes we see a tall, thin, rather pale youth, who appears to be still growing, and are inclined to say, "Give him two or three more years, and he will broaden out and become strong and healthy," until we find that his urine presents a trace of albumen. In neither case can we pass the albumen over as of no consequence. One must not spare the time and trouble required for the investigation, both microscopic and chemical, of such cases, but busy practitioners rightly make use of one of the institutions which are open to us for having an accurate examination made, with all modern appliances, of the microscopical and chemical characters of blood, sputum, and urine, and other pathological products. By the rules which I have given—by the presence of albumen or globulin, or nucleo-albumen, or pus, or minute quantities of blood—combined with the presence of casts, whether the small hyaline casts which are often compatible with health, or the more common granular, epithelial, fatty, or blood-casts, we can learn to put a case of albuminuria into its proper place. As I have already said, I think a great many cases in boys and young men will be found to be not physiological, nor yet examples of Bright's disease, but due either to some condition affecting the lower urinary passages, which has nothing to do with the kidneys, or else due to some weakness of the co-ordinating vaso-motor system, by which cold causes temporary cyanosis and passive congestion of the kidney.

In making a decision in these important cases when they come before us for diagnosis, or for life assurance, on what points ought we to depend?

The first thing is not to trust to one test. Every test has fallacies, and if we find a precipitate with picric acid, we ought not to be satisfied with that test, but use the boiling test, and perhaps the ferrocyanide and acetic acid test also. Those three tests are sufficient to establish the preliminary fact that it is a true case of albuminuria. The second point is never to condemn such a patient or candidate at the first interview. Its occurrence may be accidental, and one ought to see him a second time or oftener. It is again important to get the specimen which is passed after a meal, as well as that which is passed on rising in the morning; one of these specimens may show the presence of albumen and the other may not. Then comes the important question of the specific gravity. If the urine is of good colour, and is of high specific gravity, then a trace of albumen may very probably be accidental. If, on the other hand, and especially in a man who is over forty, we find the merest trace of albumen, and along with that the urine is pale and of low specific gravity, then we look carefully for evidence of increased tension in the radial artery, and we listen carefully at the heart to distinguish the first signs of ventricular dilatation or hypertrophy, and we inquire very carefully as to the applicant's habits and occupations. When our patient or candidate is under twenty-five—even if the quantity of albumen is very small—and we find it twice, I do not think we can recommend him for life assurance. The right thing to do for him, and the right thing for the office, is to put him off for a time. If the candidate is wise, he will go to his family doctor and tell him; he will ascertain the cause of the proposal being deferred, and will take the young man under treatment. In a large number of these cases the result is that they are perfectly restored to health.

In such cases the most important thing of all, I think, is to keep the surface of the body warm; to see that the subjects of this condition are clothed in wool from the neck to the feet. We know boys are apt to be careless in this respect, and schoolboys particularly so. The second thing is to forbid bathing in the open air, or wherever the boys are tempted to stand about instead of at once rubbing themselves down and getting their clothes on. The next point is to restrict the amount of meat, and to entirely prohibit all stimulating liquors, though happily in modern times the majority of boys are brought up without beer or wine. When albumen continues it is very beneficial for the patient to have a hot bath before going to bed. That will often prevent the morning albuminuria which, although not indicative of organic disease, is certainly in many cases a precursor of it. At the same time it is important to keep the bowels open; the effect of a regular action of the alimentary canal is undoubtedly to ease the kidneys. As to drugs, the only one I have seen of real value is perchloride of iron. These lads are generally pale as well as thin, and they improve rapidly under the administration of the iron. If the parents can afford it, it is desirable

Digitized by Google

that the youth should spend the next winter in the south of Europe, or on the south-west coast of Devon or Cornwall, or in the Channel Islands. Where that cannot be managed, if you have a patient who is intelligent and will follow out your advice as to clothing and feeding, and abstinence from chills, and taking no liquor, it is astonishing how many of these cases will get perfectly well. Then, in eight or twelve months from the first examination for life assurance, they can appear again, and you will be able to insure them with a good conscience. On the other hand, I must confess that more than once I have noticed youths who came thin and pale, and with a little albumen present in the urine, who did not develop Bright's disease, but who never became robust, healthy men, and in several cases finally developed tubercle. Therefore one should be very careful in such cases not to minimise the significance of the albuminuria, but to recognise that it shows a certain vulnerability of constitution.

The old plan of sending every consumptive patient on a voyage was often a mischievous one, for we must remember that those who run across the sea do not always change their mind or their body either, and that a perfectly salubrious climate does not exist on this earth. So I do not think we must promise certain benefit from living abroad to those of our patients who are rich and able to go to any climate we recommend, or need commiserate those who are obliged to stop in this country to do the best they can under our advice. I think you will bear me out that, as in cases of phthisis, so also in albuminuria, some of our best cases have been amongst our poorer patients, in hospital or otherwise, to whom it would be cruel to prescribe leaving the country, or very often even leaving the town or district in which they live, and who nevertheless by care, and regimen, and proper clothing, and regulations as to drink, do finally reward our efforts by recovering. On the other hand, one has known people who had every opportunity, and who yet unfortunately have succumbed to Bright's disease. I remember a patient living in the north of England, a wealthy man, who had Bright's disease, and asked me what he should do the next winter. I told him that of all the places he could go to, probably Egypt was the best. He went there, and for a time enjoyed the sun and the dry, clear air very much; but I afterwards heard from his brother that he went out one day when a cold wind was blowing, after there had been some rain, caught a chill which ended in pleurisy, and so he died, just as he might have done if he had stayed in England. Since the British have obtained possession of Egypt it is said that we have taken our climate out there; and it seemed that in this instance it was true. On the whole, however, increasing experience has given me increasing hope, not only of relieving, but of curing patients suffering from albuminuria. Not only in the earliest stages of Bright's disease, but even in advanced cases, it is wonderful how much power of recovery is sometimes met with. At one time the best authorities believed that chronic albuminuria was "as fatal as phthisis." Now I believe we may say that it is *curable* as phthisis.

To Reduce the Alkalinity of Urine.—Attention has been called by R. Hutchison, in the 'British Medical Journal,' to the value of acid sodium phosphate as an acidifier of urine. According to this author it has the greatest acidifying power of any preparation employed. It is a valuable agent in the treatment of cystitis, accompanied by the use of urotropin, which produces its best results in acid urine. The foregoing preparation is readily soluble in water, and may be given in doses of 30 to 60 grains every three or four hours if necessary.

Adrenalin and Atropin.—R. Mengelburg, in 'Annals of Ophthal.,' lays great stress on the fact that a combination of adrenalin and atropin is liable to produce symptoms of atropin poisoning, even in very small quantities. In several cases, although no idiosyncrasy to atropin was present when given alone, marked effects were produced by combining the two. His explanation is that through adrenalin the vessels of the mucous membrane of the canaliculus are much contracted, and consequently the mucous membrane is made much thinner and the canal more patent, and in this way a larger opening is made, through which the atropin can pass into the tear-duct and from thence into the nose. He therefore advises extreme caution in using this combination.—*Journ. Amer. Med. Assoc.*, November 21st, 1903.

Digitized by Google

THREE CLINICAL LECTURES
ON
HEAD INJURIES.

Delivered at the Medical Graduates' College and Polyclinic.

By T. P. LEGG, F.R.C.S.,
Assistant Surgeon, Royal Free Hospital; Surgical Registrar and Tutor, King's College Hospital.

LECTURE I.

Injuries of the Scalp and Face.

Gentlemen,—My first duty is to thank the Council of this college for the honour of being asked to deliver a course of three lectures before such an audience as this, composed of graduates in medicine and surgery. A difficulty at once arose in the choice of the subject to be discussed. On looking over the lists of previous lectures I found that whilst the diseases of the human body had been very fully dealt with, injuries had been scarcely touched upon. I have chosen as my subject injuries of the head, because there are none more important both from the diagnostic and prognostic point of view. Patients who recover from the immediate effects of the accident may, many months or years afterwards, suffer severely and sometimes intractably from the remote effects.

Importance of slight injuries.—A very slight superficial injury, perhaps only a scalp wound, may be accompanied by extensive damage to the bones of the skull or to the brain itself. It is only by attention to what perhaps may appear to be trivial symptoms, such as a confused manner, indistinctness of speech, or general irritability of temper, that a correct diagnosis may be made or suspected, and the patient treated accordingly. It is in these cases, especially when there is an alcoholic element present, that most mistakes in diagnosis are made, usually with disastrous consequences. It is these patients whom one has the greatest difficulty in persuading of the necessity of being kept quietly in bed for twenty-four hours or more before one is able to say that some serious injury has not been sustained by the brain or the other intra-cranial contents. As an example of this difficulty and of the importance to be attached to slight symptoms, the following is a typical case:—An elderly man, otherwise quite healthy, was knocked down by some roughs and brought to the hospital. He was quite conscious, and walked into the receiving-room. I found that there was a dirty scalp wound, but no evidence of any fracture of the skull. He was somewhat dazed and indistinct in speaking. He was placed on a couch and told to stay there till I gave him permission to go. However, he got up and went on his own account whilst I was away. When he got home he sent for his own medical man, and in the course of a few hours he rapidly became unconscious. Trephining was performed without relief. Post mortem an extensively lacerated brain and fracture of the base of the skull was found. It was the mental condition which made me suspect that there was something more serious than the scalp wound in this man.

I have said twenty-four or forty-eight hours, because in most cases, symptoms and signs pointing to severe intra-cranial lesions will have come on in this time. This is not always the case; there are many exceptions—as, for example, this case, reported by Russell and Johnson in the Clinical Society's 'Transactions,' vol. xxxiv (1901). A man æt. 33 fell down a flight of stone steps; he became unconscious and remained so till next morning. He then walked to a doctor's, who dressed a superficial scalp wound in the left parietal region He complained of headache and of being shaken by the fall. During the next five days nothing noticeable happened; on the sixth day convulsions, involving the left side of the body and spreading across to the right side of the face and the right leg, began and were frequently repeated. An irritative lesion of the right motor cortex was diagnosed, and the patient was trephined over the right motor area. Several drachms of blood were evacuated on incising the dura mater. The brain appeared normal. The man made an excellent recovery, having no fits after the operation. There was a strong family history of epilepsy, but the conditions found and the results of the operation disprove that the case was one of ordinary epilepsy.

Anatomical considerations.— To understand the full consequences of injuries and why the different results follow, it is important to bear in mind the anatomy of the head. We may place the cranial structures in three groups: (1) the external soft parts; (2) the bones of the vault and those of the base; (3) the internal soft parts—the brain and its

Digitized by Google

membranes. Each of these may be damaged without the others being affected.

Layers of the scalp.—The scalp consists of five different layers: (1) the skin, with its abundant hair-follicles and glands; (2) a layer of fibrous tissue and fat, generally called the dense connective-tissue layer, which is intimately adherent to both the skin and the aponeurosis of the occipito-frontalis. It contains the blood-vessels, which run in fibrous sheaths or canals which are adherent to the outer wall of the vessels. When these are wounded they cannot retract within their sheaths, and as their walls are unable to contract they bleed freely; and owing to the rigidity of the fibrous tissue, it is not easy to pick up a bleeding vessel in the scalp, but any hæmorrhage may be easily stopped by pressure against the underlying bones; (3) the aponeurosis of the occipito-frontalis. This is a most important layer of the scalp, for two reasons—(*a*) if it is not wounded or divided in a scalp cut, this will not gape; (*b*) if it is wounded the cut not only gapes but another layer; (4) the loose connective-tissue layer is opened up, in which, if hæmorrhage or infection takes place, wide-spread mischief may result. This loose connective space extends from the supra-orbital ridges in front to the superior curved line of the occipital bone behind, and on each side as low as the zygomatic arches, beneath which it is continuous with the areolar tissue of the zygomatic fossæ. It is the presence of this layer which allows the movements of the scalp to take place, and a flap to be turned down either for an operation or as a result of an accident. The main blood-vessels of the scalp are superficial to this layer; hence the flap rarely sloughs, and it can thus be always replaced in position, provided care is taken to drain this "dangerous area." The last layer of the scalp is the pericranium, which covers each individual bone separately, being only slightly adherent to them, and being continuous with the dura mater at the periphery of each, along the lines of the sutures. The pericranium differs from the periosteum of the long bones in two important respects: it does not supply the bones with nutriment to any extent, and it has no bone-forming properties. Hence, when it is stripped off, as it often is, in injuries and in trephining, the bone does not die, the blood being obtained from the diploë and the dura. Or if necrosis should occur it is the external table only which dies as a rule, this being less freely supplied with blood. In the repair of fractures callus is not formed; if any bone is removed a gap will permanently remain, its edges being smoothed down and rounded off.

When the skull is examined two things are at once evident: the varying thickness of the individual bones, and the fact that those bones which are thickest and densest are placed at those parts which are most exposed to injury, to afford a maximum of security to the parts of the brain beneath, which are the most vital portions of the central nervous system. Thus the medulla is placed on the basi-sphenoid and basi-occipital, which afford it ample protection from all but the most violent injuries; the Rolandic fissure, around which the motor centres are grouped, is protected by the parietal, the thickest bone entering into the formation of the vault; the cerebellum is protected by the strong petrous and occipital bones, the latter of which is especially dense in the middle line posteriorly and around the foramen magnum. whilst the lateral thinner parts are covered by the muscles attached to it. Similarly the thin squamous portion of the temporal is protected by the temporal muscle. At the anterior end of the skull strength is provided by the thick supra-orbital ridges and external angular processes. The thickness of the skull bones is due to the diploë, which contains the blood-vessels in large canals, from which emissary veins lead to the sinuses in the dura and to the veins of the scalp on the outside. Hence, if after an injury the wound becomes infected, extensive thrombi may form in the diploic veins, leading to the septic osteomyelitis, which may be local or general. The large sinuses may also be involved by direct extension of the septic thrombus; and the clot, becoming detached, may be carried into the general circulation, leading to septicæmia or pyæmia.

The sutures are important, because they serve to break up and interrupt any violence applied to the bones, and, moreover, they are sometimes mistaken for fractures; their position and serrations* will serve to prevent this mistake being made. Sometimes the sutures are started, as a result of violence. Under these conditions it is probable that more or less severe injuries will have been

* These become less marked with advancing age.

Digitized by Google

sustained by the intra-cranial contents, and the starting becomes of less importance. It is a rare accident in children. As age advances the sutures become obliterated and the bones synostosed. With advancing age the bones become less elastic, which, together with the increasing liability to accidents, explains why fractures are more frequent and extensive in adults than children.

The coverings of the brain.—The dura mater is so intimately adherent to the base of the skull as to be only separable with great difficulty. Over the vault this attachment is much less intimate, and in the adult it can be easily stripped off the internal table; in children it is so closely adherent to the vault that it can only be separated by the exercise of considerable force. Being a thick tough membrane, the dura protects the brain beneath, and, having a rich vascular supply, it forms the chief source of nutriment to the bones. As it is separable without great difficulty from the vault, a blow may detach it, and blood or pus may accumulate with rapidity between it and the bones. Owing to its toughness considerable violence may be applied to it without perforation taking place, and consequently the subdural space is not opened. Its chief vessels being on the outer side, when these are torn the blood accumulates beneath the bone, extending as far as the sutures, where the dura is continuous with the pericranium, which serves to limit the hæmorrhage unless these connections are torn. Moreover the arteries are more closely connected with the dura than with the bones. It is important to remember this, because in operating for the arrest of hæmorrhage due to rupture of the middle meningeal artery, the torn end of it will often be found lying on the dura, away from the bone, where it is not always easy to pick up and tie. The relation of the venous sinuses to the dura is important; they are only channels in it, and, when injured, blood may escape either into the subdural space or beneath the bones, or into both positions at the same time. The blood-pressure in them being low, if there is an external laceration the signs of compression will come on slowly; on the other hand, from a laceration on the inner surface wide-spread extravasation may result in the subdural space.

The space between the dura and the arachnoid is in most parts a narrow capillary cleft containing but little fluid, the cerebro-spinal fluid being lodged in the subarachnoid space. This space is larger and more marked in some parts than in others, especially at the base of the brain, where it extends in front over the pons and interpeduncular space as far forwards as the optic nerves. Posteriorly there is a large space between the medulla and the under surface of the cerebellum. The cerebro-spinal fluid here collected forms a further protection to the vital centres situated in the medulla. In the longitudinal fissure just above the corpus callosum and below the falx there is another space. Through the foramen of Majendie, an opening in the pia mater covering the fourth ventricle, the fluid in the lateral ventricles of the cerebral hemispheres is continuous with that in the subarachnoid space, which is in direct continuity with that in the spinal canal. Thus we have the explanation of the large amounts of fluid which may be lost in cases of fracture of the skull, if the dura and arachnoid are torn, and of the tumour—the traumatic cephalhydrocele or false meningocele—which occasionally forms in children after fractures of the vault of the skull.

The pia mater is the vascular investment of the brain, which it closely covers in all parts. When injured wide-spread extravasation results on the surface, and the brain itself is frequently damaged at the same time.

Scalp wounds and their effects.—Let us take a simple injury first. A patient receives a blow or falls on his head. In either case a clean cut may result, owing to the tenseness of the scalp. The aponeurosis may or may not be divided; on this the prognosis largely depends. The superficial extent of the wound is not very important. The immediate danger is hæmorrhage, which may be very free, but in most cases may be arrested, at least temporarily, by pressure. If one of the deep temporal arteries is wounded, it may be necessary to enlarge the wound. If the wound is disinfected it may be sutured. By disinfection is meant not merely washing with some antiseptic, but very often cutting away the edges of the wound to remove the dirt, which is often ground in. If "the dangerous area" is opened, a drainage-tube may be put in. If these precautions are not taken, septic complications are very likely to follow either in the skin or in "the dangerous area." In either case grave results follow, especially in old people. If the sepsis occurs in the skin, extensive œdema and cellulitis

Digitized by Google

may occur; if it occurs in the subaponeurotic space, an abscess, only limited by the attachments of the occipital and frontalis muscles, may form. The scalp will be lifted up, but it will not slough, because its blood-supply is not interfered with. A large boggy swelling forms and the bones beneath may be infected, infective osteomyelitis resulting in septicæmia, or pyæmia may come on.

Case 1.—A boy, æt. 6 years, fell and cut his head on the pavement. There was a wound three quarters of an inch long above the eyebrow, exposing the pericranium. One stitch was inserted after the wound had been washed with 1 in 40 carbolic lotion; four days later the eyelids were so swollen that he could not open them. The wound was suppurating and the stitch was removed. On the eighth day the temperature rose to 103·8°, the child was very ill, drowsy, and apathetic. Extensive œdema of the scalp and eyelids was present. Incisions were made in six places through the aponeurosis to the bone to let out the inflammatory fluid; no pus was found, only blood-stained serum. The wounds were plugged and dressed with gauze wrung out in 1 in 2000 perchloride of mercury lotion.. The child then made a rapid recovery.

Case 2.—A child, æt. 2 years, fell off a chair and struck its forehead against the fender. The next day the forehead was swollen and puffy, the child being drowsy and fretful. Two days later he vomited several times. Chloroform was administered and the wound was enlarged; gauze drains were put in; the temperature was 102°. No improvement following, six incisions were made over the left side of the scalp, each being made parallel to the direction of the vessels. Semi-purulent fluid escaped from the tissues of the scalp. The bone at the site of the original wound was quite bare. Each incision was plugged with gauze. The left parotid lymphatic glands were suppurating, and two drachms of pus were evacuated from them. Five days afterwards pneumonia developed at the bases of the lungs. Subcutaneous abscesses developed over the right elbow and heel. The child died about sixteen days after receiving the injury. At the post-mortem the left lung had numerous petechiæ between the upper and lower lobes and on the posterior surface of the lower lobe, which was studded with numerous minute abscesses. The liver was pale and softer than normal. The brain and meninges were normal. This is a typical case of pyæmia following a simple scalp wound, and it illustrates the points I have mentioned. The first case was one of toxæmia, and though the patient was very ill for some time, he subsequently recovered.

In other cases the inflammation may be more localised and lead to localised osteomyelitis and necrosis with an abscess, which may be intracranial, between the dura and the bone, or in the brain itself. The wound may have healed by this time. A localised œdematous swelling forms in the scalp over the abscess, and is well known under the name of "Pott's puffy swelling."

Hæmatomata.—Hæmatomata of the scalp may appear in three different situations: (1) superficial to the aponeurosis; (2) beneath the aponeurosis, the subaponeurotic variety; (3) beneath the pericranium, the subpericranial form. The two last varieties are the most important, as they are often the result of fractures, and they present special characteristics. Any one of them may be the result of a simple contusion, and, owing to the anatomical peculiarities, may give rise to difficulty in diagnosis as to whether a depressed fracture is present or not. This difficulty most often arises when the hæmatoma is in the subcutaneous tissue. The edge of the swelling is firm and hard, raised above the surrounding parts, the central portion being softer, elastic, and depressed below the margins. By firm pressure on the edge this may be flattened out, and the firm bone be felt continuous with that beneath the central portion of the swelling. Macewen has explained this effect by supposing that the central portion is the part struck and violently compressed against the bone. The cellular tissue is thus destroyed and scattered centrifugally, its elements together with the neighbouring tissue causing a heaping-up and condensation at the periphery which becomes more dense by the infiltration of blood. The soft central part is caused by the skin being separated from the aponeurosis by the fluid blood.

Subaponeurotic hæmatomata due to simple contusion are not frequent, as there are only a few small blood-vessels in this space. As a result of fracture they are common, and are characterised by their tendency to be diffused, and a "loose feel," the scalp being raised by the blood. They are thus much more important than the previous form, and they should always raise the question of

Digitized by Google

fracture and the possibility of intra-cranial hæmorrhage.

The following case will illustrate these points: A youth, æt. 18 years, was admitted to the hospital quite conscious, after a fall from a horse on to his head. Soon afterwards he vomited and became somewhat drowsy. The left pupil was larger than the right; pulse 72; over each parietal bone there was a well-marked hæmatoma with raised edges, giving a sensation of a depressed fracture. Both of them were elastic and felt solid. The next day he was irritable and restless, lying in an attitude of general flexion. Slight facial palsy on the left side was noticed. Nothing of note occurred till three days later; then he had well-marked twitchings of the muscles of the left side of the face, lasting a few minutes. He was quite conscious, but seemed to be more drowsy after they ceased. During the next twelve hours five more similar attacks occurred, always confined to the face. There was no paresis of the limbs. Well-marked internal squint of the right eye followed. The question of trephining was discussed, but the operation was deferred, and no more twitchings occurred. A week later the hæmatomata were still present and tense, being of the same size. At the end of a month they had disappeared, and the patient was quite well. I think this patient had a fissured fracture of the skull, and probably a slight laceration of the brain, over the face area on the right side, with effusion of blood which irritated the face centre, and at the same time pressing on the sixth nerve paralysed it, leading to the squint in the right eye. There may have been a fracture of the base, but of this there was no evidence.

The third form of cephal-hæmatomata is most often seen in infants and new-born children. It is generally situated over the parietal bone, being localised to the bone by the attachments of its periosteum at the sutures. It is caused by injuries received during birth, perhaps by long-continued compression or the forceps. There may be a fracture of the bone, which leads to the hæmorrhage by causing rupture of vessels of diploë. The blood may be only slowly absorbed, and the swelling will then persist for a considerable time after birth. The edges become hard from the deposit of fibrin, but firm pressure shows that the swelling is not bony. A short time ago I saw an infant who had a firm hard swelling over the right parietal bone. The mother could give no history of how or when the swelling first appeared. It was diagnosed as a cephal-hæmatoma, and on being punctured clear blood-stained serum was drawn off.

The treatment of hæmatomata does not call for any special mention; the important point to remember is that they are often only an accompaniment of more severe injuries, and that they may persist for a long time, giving rise to a blood cyst, which may require tapping. If a scalp wound is present they may become infected from it and suppurate; if this should occur a free incision is required and drainage.

Injuries of the soft parts of the face.—These injuries are frequent, and result from all kinds of violence. They are of all grades of severity, from a slight cut to the formation of a large flap, involving everything down to the bone. In the summer of last year I had under my care, a little girl who had a wound of the face which involved the lower eyelid and extended through the cheek to the upper lip, turning down the whole of these structures in a flap. Both sides of the face were ecchymosed to such an extent that the child's features were unrecognisable. The wound was carefully disinfected and sewn up, a drainage-tube being inserted. Especial care was taken in suturing the lower eyelid at the inner canthus to accurately appose the edges of the wound, which healed almost without any suppuration; the lower lid was only slightly drawn downwards. I have seen this child recently; the scar is evident, but the eyelid is not everted and there is no epiphora. In connection with these injuries three things are important: (1) the hæmorrhage; (2) the depth of the injury and the deep structures which are damaged; (3) the scarring and the resulting deformity. Punctured wounds are thus the most dangerous.

1. The hæmorrhage is often great, and may be dangerous, especially when such a vessel as the facial or one of the deep temporals is wounded. It is often necessary, when one of these vessels is wounded, to enlarge the wound in order to tie the bleeding point to permanently arrest the hæmorrhage. Temporarily it may be stopped by firm pressure. Stabs in the parotid region and behind the angle of the jaw often lead to free bleeding. The blood simply wells up, coming from the numerous veins of this part, so that it is impossible

Digitized by Google

to tie all the bleeding points. Firm pressure applied by gauze plugging is usually sufficient to arrest the hæmorrhage. In wounds of the lips the coronary arteries bleed freely, but the bleeding is easily arrested by compression of the lips or by merely bringing the margins of the wound into apposition by sutures. In the other parts of the face the hæmorrhage is readily arrested by ligation or pressure. In any of these injuries extensive ecchymosis is likely to follow, but is unimportant. Moreover, on account of the freedom of the blood-supply, sloughing is unlikely to occur. For the same reason healing readily takes place after suturing the margins of the wound.

2. *The depth of the injury.*—When the importance of the structures beneath the soft parts of the face is considered, the necessity of discovering the depth of the injury is at once apparent. For example, the antrum of Highmore may be penetrated; through a wound in the eyelid or up the nostril the brain may be involved; in the parotid region the facial nerve may be divided. The instrument which inflicted the injury may be retained in part or altogether. The superficial part of the wound may heal completely, and the foreign body remain quiescent. Or it may cause extensive cellulitis, either localised or diffused. If such a foreign body is not retained, suppuration may still follow, as the instrument causing the injury is often infected. Further, as a result of the sepsis, secondary hæmorrhage may come on at a later date, or the infective process may spread to the meninges through the veins, leading to septic meningitis.

The actual damage to the parotid gland itself does not lead to a permanent fistula. For a time its secretion may escape through the wound, but unless the main duct is damaged the fistula closes. If the main duct is injured, a permanent fistula may follow, requiring a plastic operation for its closure. The capsule of the gland and the separated lobules should be united by sutures before the skin incision is closed.

The facial nerve may be damaged before or after its division. In the latter case there is not much hope of being able to find the divided ends to join them together. If the main trunk is severed by a stab in the parotid region, before its division, an attempt may be made to unite the ends by enlarging the wound and searching for the nerve behind the ascending ramus of the lower jaw and below the lobule of the ear.

3. *The scarring and the resulting deformity.*—I have already pointed out the deformity which sometimes follows injuries of the eyelids. In the lips unsightly deformities also result unless care is taken to sew up wounds in them accurately. The stitches must be passed deeply to obtain a good hold of their whole thickness, and it is especially important to bring into apposition the line of junction of the mucous membrane and skin. If the whole thickness of the lip is not brought together a V-shaped depression or notch results. Interrupted silkworm or horsehair sutures are the best to use. The cicatrices following deep burns are the most disfiguring and disabling of all scars. They frequently extend on to the neck, interfering with the movements of the head on the neck; they cause wry-neck; they draw down the angle of the mouth and the eyelids; and, moreover, are not easily rectified. Numerous plastic operations are generally required to improve the patient's appearance.

Fractures of the bones of the face and jaws.—These fractures are among the most frequent of head injuries, and are important: 1st, because great deformity is liable to result if treatment is not carefully carried out; 2ndly, they are often compound into the mouth as well as externally; 3rdly, septic complications leading to necrosis are not infrequent; 4thly, interference with function.

Isolated fractures of the malar bone and zygoma are rare. They are generally combined with fracture of the superior maxilla, of the orbital walls, or the base of the skull. When the patient is seen soon after the accident the diagnosis is easy; as a result of the hæmorrhage marked swelling soon appears and obscures the fracture, making the diagnosis difficult. The ecchymosis extends into the orbit and may cause displacement of the eyeball.

A man, æt. 52 years, fell and struck his face against some railings. There was a lacerated wound just below the left malar bone, which was broken; the anterior surface of the superior maxilla was fractured, the antrum being opened. His eyeball and vision were unaffected. Under an anæsthetic the fragments were placed in position, bruised portions of tissues were removed, and drainage-tubes inserted. The next day very ex-

Digitized by Google

tensive ecchymosis of the cheek had appeared, extending into the orbit beneath the conjunctiva; there was no proptosis. Some suppuration occurred, but without any necrosis. No deformity resulted, and there was no interference with the movements of the lower jaw.

Fractures of the lower jaw.—The commonest seat of this injury is in the body near the canine teeth; when great violence has been applied the fracture may be double. The alveolar margin alone may be involved. Fractures about the angle and ascending ramus are not very infrequent, but fractures of the coronoid process and condyle are quite rare. When the body is broken unilaterally close to the symphysis, the displacement may be only slight; if the line of fracture through the bone is oblique, overlapping of the fragments is common. When the lines of fracture are symmetrically placed on each side of the symphysis, the anterior or middle fragment is drawn downwards and backwards by the action of the supra-hyoid muscles. Fractures of the ascending ramus usually lead to no displacement, because the fragments are held in position by the internal pterygoid and masseter muscles. If the body is broken in front of the masseter, the posterior fragment is drawn upwards and forwards by the temporal and masseter muscles, the anterior fragment being depressed and carried downwards by the digastric and hyoid muscles. If the condyle is broken the body of the jaw is carried over to the side of the fracture, the condyle itself being drawn inwards and forwards by the external pterygoid.

Fractures of the body and alveolar margin are invariably compound from laceration of the mucous membrane, and not infrequently there is an external wound in addition. Necrosis is a frequent result, and is sometimes extensive. Delayed union is common; non-union is rare. I have recently had under my care a man who about Christmas, 1902, had broken his jaw. There was a suppurating wound in the middle line of the chin, leading to the fracture, which was just to one side of the symphysis. Very little deformity was apparent; there was no attempt at union, and crepitus was easily obtained. The abscess was drained. At the present time, ten weeks after the accident, there is still no union. Crepitus is as a rule easily made out, if the fingers of one hand are placed on the inside of the jaw, and those of the other on the outside. It is not uncommon to see patients who have received a violent blow on the side of the face with a large hæmatoma in the masseteric region. This should always suggest examining the jaw in this manner for a fracture, which is not infrequent, and which otherwise would be missed. In fractures of the body when the deformity is slight, crepitus can be felt by placing the fingers on the teeth and the thumb below the inferior border; by using both hands, one on each side, the abnormal mobility and crepitus can be felt generally quite readily. Severe pain, which is common to all forms of fracture of the lower jaw, and inability to open the mouth properly, would emphasize the need of making such an examination. The hæmorrhage is not usually great, and ceases spontaneously. In one case only have I seen any serious result, apparently due to it, follow. This was the case in a strong, healthy young man who was admitted to the hospital with a fracture of the body of the jaw in two places, and who also had a compound fracture of the lower limb, for which amputation was required in the lower third of the leg. The hæmorrhage from the jaw persisted, the patient dying within twenty-four hours. There were no other injuries, and at the post-mortem nothing more was found to account for his death. In the treatment of these injuries many methods have been devised, the form of splint most frequently used being Hammond's interdental wire splint. As it is essential that the splint should fit accurately, it is advisable that the aid of a dentist should be sought in making it. This splint cannot be readily applied to the teeth when they are close together. I here show you a cast of the teeth of the lower jaw, showing how close they are to one another, and how impossible, or almost so, it would be to use a Hammond's splint. In such a case, and perhaps in most cases, a Hayward's or Kingsley's splint is the best to use. This splint consists of a vulcanite plate, which is moulded to the teeth. To it are attached two metal bars, one on each side, and passing backwards over the cheeks from the angles of the mouth. It is kept in position by a bandage passing from one bar to the other beneath the chin.

When there is not much displacement, an ordinary four-tailed bandage, with or without a trough splint, is enough. As a primary method of treatment wiring the fragments is not often required;

Digitized by Google

when necessary the fragments should be drilled from a wound made below the chin rather than from inside the mouth. In this way the wires are placed altogether outside that cavity, and a source of infection is avoided. Union takes place in four or five weeks.

An essential point in the treatment of all cases is to keep the mouth as clean as possible; the frequent use of a tooth-brush, wiping the mouth out thoroughly after all food with an efficient antiseptic, is necessary. For the first few weeks all nourishment must be given in liquid form, preferably by a long tube passed into the mouth through a gap between the teeth or behind the last molar. Loose teeth should not be removed, as they become fixed again. It is important to see that teeth are not impacted between the fragments. Any abscess is to be opened and drained; these collections of pus often point at the inferior margin of the body, and are best opened from the outside. Necrosed bone is removed as soon as it becomes loosened.

Fracture of the superior maxilla is always due to severe direct violence. The extent of the fracture varies from a small part of the alveolar margin being torn off, to complete comminution of the whole bone, together with the neighbouring bones. Some time ago I saw a policeman who had both jaws so completely comminuted that one could pass the finger through the antra into the naso-pharynx and nose. The nasal bones, malar and ethmoid, were all smashed up, and the infra-maxilla was broken as well. Needless to say, the skin of the face was extensively lacerated; he was bleeding profusely, and died soon afterwards. Post mortem the base of the skull and brain were uninjured. The skin is not infrequently torn and lacerated; hence the fracture is often compound in two directions—into the mouth and externally. Suppuration is frequent, and provision must be made for free drainage.

It is impossible to describe these fractures separately, as each case differs from any other. Certain general facts are, however, important. First, when the body of the bone is involved, great deformity of the face is likely to follow, unless the fragments are replaced. This is best accomplished under an anæsthetic, manipulating them into position, making an incision, if necessary, through the cheek, the scar being less unsightly than the deformity resulting from non-reduction of the fragments. Second, owing to the vascularity of the bone, necrosis does not take place; therefore loose portions should not be removed, because they will unite and greatly reduce any deformity. Third, hæmorrhage is sometimes free, the blood coming in part from the terminal branches of the internal maxillary which run in canals in the bone. Pressure or crushing the open ends of the canals is sufficient to arrest it. When the floor of the orbit is involved, exophthalmos may occur. Secondary hæmorrhage also appears to be a somewhat frequent result. Fourth, fractures involving the alveolar margin are accompanied by loosening of the teeth; these should never be removed; even those which have fallen out, if replaced at once, may become firmly fixed again. In the treatment of these fractures it is most essential to restore the outline of the teeth again. To do this some form of dental splint is required. This may be fixed to the lower jaw, which is then brought up against the upper teeth, the two being kept in apposition by bandaging. Or an interdental splint may be applied directly to the alveolar margin. Fifth, extensive comminution of the bone is likely to be accompanied by other injuries, such as fracture of the base and intra-cranial injuries, which are themselves fatal. Sixth, the lachrymal apparatus may be affected; if the nasal duct is torn across, permanent epiphora may follow from stricture of the duct. Surgical emphysema may also occur, and any wound of the skin is to be carefully sutured, especially in the neighbourhood of the inner canthus, to prevent deformity from contraction and epiphora due to the eversion of the lower lid. Seventh, the infra-orbital nerve may be crushed, leading to neuralgia or numbness of the cheek. The branches of the facial may be torn, so that some loss of motor power in the face may follow. In the treatment of these fractures the same lines must be followed as were described in the treatment of fractures of the lower jaw.

LEUGEMANN ('Deutsche Med. Woch.,' No. 23. 1903) considers the operative treatment of Dupuytren's contracture not always satisfactory, so he has tested the effect of thiosinamin. The results were good. The treatment consisted in injections into the neighbouring tissue of Thiosinamin with Glycerin and distilled water, at first daily and later weekly.—*Interstate Med. Journ.*, November, 1903.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 582. WEDNESDAY, DECEMBER 23, 1903. Vol. XXIII. No. 10.

CONTENTS.

PAGE

*A Clinical Lecture on Ozæna or Atrophic Rhinitis. By LAMBERT LACK, F.R.C.S. 145

*A Clinical Lecture on Broad Ligament Tumours. By ALBAN DORAN, F.R.C.S. 149

*Three Clinical Lectures on Head Injuries. By T. P. LEGG, F.R.C.S. LECTURE II.—Fracture of the Vault and Base of the Skull. 153

** Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A LECTURE
ON
OZÆNA OR ATROPHIC RHINITIS.

Delivered at the Polyclinic.

By LAMBERT LACK, F.R.C.S.,
Surgeon to the Throat Department, London Hospital.

GENTLEMEN,—Ozæna is well worthy of study on account of its importance, its common occurrence, and its exceptional pathological interest. The disease is of importance in that, although it presents no danger to life, it prevents its unfortunate victim from associating with his fellows in friendship, and even in business. It thus produces severe mental depression and bodily ill-health. The interest of the affection is shown by the number of theories which have been invented to account for it, and its intractability by the numerous methods of cure, all of which are infallible until a new one is discovered. The disease has been known from the most ancient times, being described by Hippocrates, but until quite recently its causation and pathology have remained obscure. The object of this lecture is to clear up some of the confusion which at present enshrouds this subject, to present a theory which will at any rate serve as a sound working hypothesis, and to base upon it a rational method of treatment.

Definition.—Ozæna may be defined as a disease of the nose, associated with an intensely *fœtid* muco-purulent discharge in the form of large *crusts*, with undue width of the nasal fossæ, and with changes of an atrophic nature in the nasal mucous membrane. This definition is imperfect, as every symptomatic definition must be, for the symptoms are by no means constant in every case. Thus in rare cases the fœtor may be slight, although the other symptoms are well marked; sometimes the nasal fossæ are not especially wide; and more or less atrophy of the mucous membrane may exist apart from the other symptoms.

Digitized by Google

Before discussing the various theories of the affection, I wish to draw your attention to some of the more salient features of the disease. The following analysis is based upon the records of 133 cases of ozæna, in all of which the prominent symptoms were well marked.

1. *Sex and age.*—Seventy per cent. of the patients were women. In 39 per cent. of cases the affection commenced under the age of five years; in a further 35 per cent. under the age of twelve. The majority of authors state that the affection usually appears at or about fifteen to seventeen years of age. These estimates are based upon patients' statements with regard to the commencement of the evil smell and crusts, but if close inquiry be made it will frequently be found that the *discharge* commenced several years previously. Thus Bosworth states that atrophic rhinitis is a disease of adolescence, but that the purulent rhinitis, which in his opinion is a constant precursor of ozæna, is a disease of childhood. It seems to me more rational to date the commencement of the disease from the first appearance of the discharge, for, as will be seen later, the other symptoms of the affection are secondary to this. In my opinion, the disease almost always commences under twelve years of age, but when it is evidenced only by a purulent discharge the majority of hospital patients are apt to neglect it, and only apply for treatment when the affection has developed to the stage of large crusts and fœtor. Typical well-marked cases of ozæna in infancy are, moreover, not uncommonly met with.

2. *Cause.*—In the great majority it is impossible to determine the cause, the disease commencing so long before the patient comes under observation. Sometimes ozæna is ascribed to measles, scarlet fever, and other severe infections, more often to severe or repeated colds. In a few cases the disease may with great probability be ascribed to the purulent rhinitis of hereditary syphilis. As will be seen later, a condition of the nose indistinguishable from atrophic rhinitis may result from tertiary syphilis in adults.

3. *Physiognomy.*—A typical facies is met with in about 40 per cent. of cases. When typical, the face is broad, the cheek bones high, and the nose in profile seems set back in the face, scarcely rising above the level of the cheeks. The nose is broad, the nostrils round, and looking forwards as well as downwards. The bridge of the nose is straight, not saddle-backed as in inherited syphilis, although this condition may also be met with. In other cases the nose is simply large and broad. This physiognomy is almost invariably associated with a roomy nose, and, as will be shown later, congenital undue width of the nasal fossæ is a strong predisposing factor to the disease. The inheritance of this peculiar physiognomy is probably the reason that ozæna has been considered hereditary. The most elaborate measurements have been made of the head by some who consider it of great etiological importance. It has been found that the majority of patients belong to the brachicephalic type, and further, that the relationship between the length of the nose, measured along its floor, and the depth of the post-nasal space, is altered, so that the length of the nose is comparatively less and the depth of the post-nasal space comparatively greater. All these measurements, however, may be summed up in the one fact that in ozæna the nasal fossæ are unduly wide, and that congenital width of the nasal fossæ is usually associated with proportionately increased breadth of the whole head.

4. *The examination of the nasal fossæ.*—The inferior meatus of the nose is invariably wide, but the middle meatus varies much. The inferior turbinates are usually small and shrunken; they may be very small, forming mere ridges difficult to distinguish along the outer wall of the nose. The middle turbinates occasionally share in the atrophy; most commonly they are normal; in a few cases they are hypertrophied.

5. *Examination of the accessory sinuses.*—Suppuration in one or more of the accessory cavities of the nose was found in 10 per cent. of my series of cases. The sphenoidal sinus and the antrum were most often involved; in two cases the ethmoidal cells were affected. This is in accordance with the general experience, but quite contradictory to the theory of Grünwald, who states that ozæna depends upon a focal suppuration, generally an affection of one of the accessory cavities. The results obtained by the method of treatment about to be described will be shown absolutely to exclude sinus disease in the large majority of cases.

6. *Histological examination of the mucous membrane.*—In ten cases small pieces of mucous membrane from the inferior turbinates were removed

Digitized by Google

and submitted to microscopical examination. In all similar changes were found. The most striking fact was the complete transformation of the epithelium—normally columnar-celled and ciliated—into squamous, non-ciliated epithelium. The underlying mucous membrane was thinner and more fibrous than normal. The racemose glands were partially degenerated and fewer in number; the vascular sinuses for the large part had disappeared. In all sections the bone itself appeared to be normal. Wingrave has investigated this subject with similar result.

7. *Relationship to tubercle.*—A marked history of tubercular disease in the family was met with in 30 per cent. of cases, and four patients had phthisis when they came under observation. This subject was investigated because the disease has been ascribed to struma. It does not help us much, however, for ozæna is certainly not a tubercular disease. The special tendency of these patients to contract phthisis may be due to the fact that the value of the nose as a respiratory organ is lost.

8. *Hereditary predisposition* is often very marked. In one family four children, three girls and a boy, and the mother had ozæna. In another family four sisters, one brother, and the mother were affected. In the last case the affected persons all bore a strong likeness, and had broad flat noses, whilst the members of the family who were not affected had narrow prominent noses. In these cases it is probable that it is the shape of the face and the associated undue width of the nasal fossæ which are inherited. This seems more probable than assuming that the disease is inherited and produces the peculiar physiognomy.

9. *Evidence of contagion.*—Evidence pointing to contagion was twice met with. Two children under my care, both with well-marked ozæna, were brought up together as sisters, but one child had been adopted, and was no relation whatever to the other. In the other case a niece was said to have contracted the disease by sleeping with an aunt who was similarly affected. The occurrence of several cases in a family may also possibly be due to infection.

Having now considered some of the more prominent features of ozæna, we are in a position to discuss the various theories of its causation; but before doing this let me draw your attention for a moment to the most prominent symptoms—the discharge, the crusts, and the fœtor. The two latter, the crusts and the fœtor, are symptoms of secondary importance only from an etiological point of view.

Explanation of crusts.—The discharge consists of muco-pus, epithelial cells, *débris*, and contains numerous bacteria. If the nose be cleansed and then packed so as to exclude the air, no crusts will be formed, thus proving that the discharge when secreted is fluid. The crusts therefore depend upon the drying of the discharge, and the drying occurs because the discharge is retained in the nose and exposed to the constant passage of air. Now the discharge is retained (1) because the nasal fossæ are unduly wide. This condition renders inefficient the action of blowing the nose. The wider the channel, obviously the less efficient is the expiratory blast. (2) Because of the absence of the cilia of the epithelium, which in the normal condition constantly keep the nasal secretion moving onwards towards the throat; and (3) because the secretion is more tenacious than usual. This last fact is disputed, but at any rate as the drying process proceeds the discharge becomes more and more sticky.

Explanation of the fœtor.—Just as the discharge when secreted is fluid, so also it is non-fœtid. The fœtor forms subsequently and is due to decomposition of the crusts, which, remaining in the nose for long periods, are infected by the bacteria in the air. Much trouble has been expended on explaining the peculiarities of the fœtor. The ozænic discharge has certainly a pathognomonic and very remarkable odour, but in this respect it is analogous to many other secretions. Thus the odour of a fœtid otorrhœa is equally characteristic, and the secretions of different parts of the body—for example, of the axillæ and the feet—have characteristic odours. The cause of the peculiarity of the fœtor in ozæna has been ascribed to the breaking up of fatty acids in the secretion, to its admixture with nasal mucus, and to the presence of a peculiar organism. In any case it may be granted that the peculiar chemical composition of the discharge either enables a particular organism to flourish, or breaks up under the influence of bacterial growth into distinctive chemical products. Let this be as it may, the important points to remember are that, just as the discharge when first secreted is fluid, so it is also odourless, and that the stench and the crusts are secondary phenomena, and require no further consideration from an etiological point of view.

Digitized by Google

There remain to be explained the source and origin of the discharge, the undue width of the nose, the atrophy of the mucous membrane.

The origin of the discharge.—The discharge probably comes from the general surface of the nasal mucous membrane, and not from any particular part. Its invariable origin from a sinus or other local source, as stated by Grünwald, is contradicted by the experience of nearly all specialists. It is, I think, conclusively disproved by the results of the method of treatment which I invariably adopt—namely, packing the nose. By this means the discharge in time may be entirely stopped. This would not occur—on the contrary, the discharge would probably be aggravated by this treatment—if it were coming from one of the sinuses. Again, the fact that the disease usually commences in early life, before the sinuses are developed, is fatal to Grünwald's views. The pathological evidence which he obtains from post-mortem examination in these cases may also be discounted. That a little mucus or muco-pus should be found in an accessory sinus post mortem may be explained by the fact that the cilia, upon which the removal of the secretion in these cavities depends, have stopped work at or immediately preceding death. A little secretion in these cavities post mortem is, therefore, of no pathological importance; disease of the sinuses should not be assumed unless there are organic changes in their lining mucous membrane or walls.

The undue width of the nose is probably congenital, and often inherited, as already stated. The importance of this factor is clearly shown by the facts (1) that the affected nasal fossæ are invariably wide; (2) that in cases of unilateral ozæna, which are by no means rare, the septum nasi is invariably deflected, so that the affected nostril is unduly wide and the unaffected nostril narrow; and (3) that in some cases polypi and ozæna have been seen to alternate,—that is, that after nasal polypi have been removed a condition resembling ozæna has supervened, and again disappeared when the polypi have returned.

The origin of the atrophy.—On this point I incline towards the theory, which was, I believe, first propounded by Bosworth. An acute rhinitis commences in early childhood, and persists because of neglect, or of general ill-health of the patient, or of the severity of the original infection. The discharge soon becomes purulent, and then, if it be examined, it will be found to contain large quantities of ciliated epithelial cells. This means that the epithelium lining the nose is constantly shed and reproduced. As the result of this rapid proliferation, in course of time the epithelium degenerates or reverts to a simpler type; the highly specialised, ciliated columnar cell is no longer formed, but its place is taken by squamous epithelium. At the same time other changes are occurring in the mucous membrane as the result of the constant irritation of the septic products. There is extensive infiltration of round-cells; the racemose glands degenerate and in part disappear; the venous sinuses, which are the most characteristic structure of the inferior turbinated bodies, are in large part obliterated. The whole mucous membrane becomes fibrous and shrunken. This fibrosis and obliteration of vessels probably hinders the development of the turbinated bone. It is doubtful if there is ever any active absorption of the bone; such changes have never been seen microscopically. On the other hand, the bone certainly does not develop, and it is quite possible that the age of onset of the disease may be approximately estimated from the size of the inferior turbinates.

This theory, I have little doubt, is the true explanation of the large majority of ordinary cases of ozæna, but a similar change may be produced in the mucous membrane as the result of ulceration in tertiary syphilis, etc. Though the changes in this case occur far more rapidly, the result is practically the same. In consequence of gummatous infiltration and ulceration the bone may necrose and come away as a sequestrum, the mucous membrane may be destroyed and replaced by fibrous or scar tissue, and the ciliated epithelium by squamous cells. Thus, as it were, at one stroke, as the result of extensive ulceration, the same changes may be produced as result from purulent rhinitis of long duration.

To sum up, then, the main pathological features of the disease. A severe nasal catarrh in childhood goes on to purulent rhinitis, with excessive desquamation of epithelium. This results in replacement of the ciliated epithelium by squamous cells, atrophy of the mucous membrane, and arrested development of the bones. Owing to the undue width of the nasal fossa, and to the absence of the ciliated epithelium, the discharge is retained in the nose, dries and forms crusts, decomposes and gives rise to fœtor, and the irritating products of the decomposition set up fresh discharge, which again dries, decomposes, etc., and thus a vicious circle is established.

Treatment.—A rational treatment must be founded upon the pathological conditions as above described. The important point to be aimed at is to cleanse the nose thoroughly and to keep it clean. The nose must be very carefully syringed. Any solution may be used; salt and water, a teaspoonful to the pint, is as useful as anything. It is not a question of antiseptics, but of mechanical removal of the discharge. The solution is best injected into the nose with a rubber syringe. Having washed the nose out, it is well to inspect it and to detach or to syringe away any crusts that remain. After this it is a useful plan to rub the mucous membrane all over with pledgets of wool on a probe, as in this way thorough cleansing is

Digitized by Google

ensured. The most important part of the treatment is, however, the next step—namely, to pack the nose with strips of cyanide gauze. A strip an inch wide and twelve to twenty inches long should be inserted, and the patient can be taught to do this for himself. This packing prevents the entrance of air into the nose, prevents the discharge drying, and prevents its decomposition. The result is that the discharge remains fluid, the nose no longer fills with tenacious crusts, and can be quite easily cleansed by simple syringing. This method is similar to that of Gottstein, who introduced rolls of wool into the nose with the idea of irritating or stimulating the mucous membrane, and so producing a more copious fluid discharge. The principle upon which his treatment was founded was wrong, and the method was painful. The use of gauze, as above described, is quite painless, is easily carried out by the patient, and is founded upon rational principles. The gauze itself inhibits bacterial growth, and yet is non-irritant. In the majority of cases it is sufficient to syringe the nose twice daily, but the packing should be worn continually, being only removed for syringing.

It may be objected that patients will not carry out this treatment, but it will be found in practice that patients like it, and that they are much more comfortable when the nose is packed. Even if it were uncomfortable, the majority of patients would do anything which promised them a hope of relief. Should it give rise to headache or discomfort at first, toleration is soon obtained by perseverance.

Again, it may be objected that mouth-breathing, which this treatment necessitates, is injurious; but in these cases it is far better for the air-passages and lungs that the air reaching them should have passed through a healthy mouth than through an extremely unhealthy nose.

Another result worthy of mention is the effect of the packing upon the ears. Many patients suffering from Eustachian obstruction and middle ear disease as the result of ozæna are markedly improved by this treatment.

And now a final word as to prognosis. These cases cannot be cured; by no known method can the mucous membrane be regenerated or the ciliated epithelium restored. But the patient's main symptoms can be entirely removed. The syringing and packing must be continued until the nasal discharge entirely ceases; it may be for three, it may be for six months. Then the packing should be tentatively omitted, but daily syringing must be continued, for the patient's nose is no longer a self-cleansing apparatus. In a case that has been so far cured that all discharge has ceased, all that remains is a dry nose, with a tendency to collect dust and mucus. If this cavity be washed out once daily with salt and water as a part of the ordinary morning toilette, no further treatment will be required and no great discomfort will remain.

A CLINICAL LECTURE

ON

BROAD LIGAMENT TUMOURS.

Delivered at the Samaritan Free Hospital for Women, October 8th, 1903.

By ALBAN DORAN, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—We have decided to deliver a course of lectures at this hospital as a means of diffusing the knowledge acquired in its wards. It is well known that our staff has not been dumb. Sir Spencer Wells did more than any of his contemporaries to establish ovariotomy as a justifiable operation, and he freely employed statistics, then of primary importance, now of far less value. Savage, C. H. F. Routh, and the present staff have written about our experiences in medical newspapers and in books, and have set forth our researches before learned societies. But in a newspaper article the author has to be taken too much at his own valuation, whilst it is only the records of deep study or original lines of treatment that are suited for societies, where they can be criticised and judged by a competent tribunal experienced in such duties. There remains much knowledge acquired within this hospital which may best be utilised by the medium of lectures.

I have chosen broad ligament tumours as my subject. They are essentially abdomino-pelvic, far more so than is an omental tumour with its lower part lying below the level of the pelvic brim, or a small fibroma of the ovary with its upper limits extending an inch or so above the pubes. Above all they have a capsule. We must never forget that the capsule is in itself an evil to the patient because it must in part be left behind, and troublesome complications long after operation may ensue.

Not many years ago a drainage-tube was inserted into the capsule after its edges had been fixed by suture to the lower end of the abdominal wound. Even now the capsule is sometimes drained by the aid of gauze. Cases thus treated often did very well indeed, but ligatures were apt to discharge from the drainage track soon after the operation—

Digitized by Google

which was better than if they had discharged elsewhere many months later. The greatest objection to drainage was the frequency of hernia at the site of the drainage track.

Hence another method has almost replaced drainage. After the tumour has been enucleated and the vessels secured with fine silk, the capsule is allowed to sink into the pelvis. The ligatures are often applied very deep down in the pelvic cavity, close to the lower bowel. It is not surprising, therefore, to find that abscesses and fistulæ not rarely develop many months or even years after the operation, even when every care has been taken to ensure perfect recovery. I have seen and known of many such cases, and on that account I now use the drainage-tube more often than I did five or six years ago.

In clinical investigation it is good to bear in mind the chief characters of the broad ligament. It is a peritoneal fold which runs along the Fallopian tube. The two layers of serous membrane meet below the tube and lie in apposition till they reach the level of the ovary. Below that level they part. Thus there is an upper and lower portion of the broad ligament.

The upper portion, or mesosalpinx, is the seat of the common, thin-walled, parovarian cyst. When that tumour does not burrow into the lower part of the broad ligament, it lies clinically outside the class of tumour of which we are speaking. It is abdominal in position and not abdomino-pelvic. True, it is encapsuled, but when the operator has enucleated it he can usually make a pedicle of the shrunken mesosalpinx, hitherto its capsule, and cut the greater part away. The patient will thus be saved from any danger such as is caused by the presence of an empty capsule in the depths of the pelvis.

The lower portion of the broad ligament begins below the attachment of the ovary. Its layers immediately part, the pelvic connective tissue, here called the parametrium, lying between them. The anterior layer is short, the serous membrane turning up very sharply on to the back of the os pubis and upwards, where it becomes the parietal peritoneum. Hence, when a tumour of broad ligament origin burrows under the anterior layer, it pushes backwards the parietal peritoneum as it rises. In consequence the abdominal portion of the tumour lies with the parietes—skin and muscles—and also the bladder in front, whilst the parietal peritoneum lies entirely behind the tumour. Hence intestine is far off, and the chances of infection of the tumour small. As a rule broad ligament tumours do not burrow forwards, but tubo-ligamentary fœtal sacs do so more frequently—a fact to be remembered in clinical research, especially as to prognosis, for gestation is likely to proceed to term, the fœtus being protected from infection.

The posterior layer of the broad ligament runs downwards, becoming the well-known fold, Douglas's pouch. When a tumour burrows under this layer it pushes up the portion of peritoneum which forms Douglas's pouch. That fold, let it be remembered, then no longer exists; its peritoneum has become the capsule over the upper part of the tumour. Below there is no true capsule, but simply the displaced connective tissue, several important structures such as the ureter, and, above all, the upper part of the rectum stripped of the peritoneum anterior to it. Hence infection of the contents of a broad ligament cyst, or of a fœtal sac with similar relations, is always possible, and hence, again, infection of ligatures after a broad ligament tumour has been enucleated is ever probable. The ligatures sometimes lie on the very wall of the bowel. As the tumour pushes up the peritoneum of Douglas's pouch it also drags up the body of the uterus on its front aspect.

These relations and their significance should be clearly borne in mind, as we cannot inspect dissections or hang up diagrams in front of patients during clinical examinations. Let us remember when we feel a mass behind the cervix that it does not necessarily lie in Douglas's pouch—for a very good reason already explained.

We will now suppose that we have patients submitted to us for examination. A woman comes with a cystic abdominal tumour. There is resonance in the epigastrium and flanks; the tumour is movable. Surely this is a simple ovarian cyst; and on bimanual palpation we expect to find the uterus quite behind its lowest part, which will just touch the pelvic brim. But no; that part lies well in the pelvis with the uterus pushed upwards and forwards. Then this must be a broad ligament cyst, we say. Not necessarily, for a simple pedunculated ovarian cyst not rarely slips in part behind the uterus pushing it up, so that the uterine body

Digitized by Google

can be felt on the surface of the tumour above the groin. The tumour can usually be pushed up, but force should never be employed; an operation is necessary whatever it may be, and then its nature and relations to the broad ligament will be revealed. If there be no burrowing into that peritoneal fold, so much the better will it be for the patient, as no capsule will have to be left behind.

Another patient consults us; she has a thin-walled tumour in the abdomen, fluctuating and flaccid. It feels like a common parovarian cyst, but on pelvic exploration its lower part is found to be deeply engaged in the pelvis, displacing the uterus laterally, forwards and upwards. The smaller the tumour is the more will the lateral displacement of the cervix uteri be marked; the larger it is the more will the uterus lie forward and upward. This is the rule* with all broad ligament tumours, for evident mechanical reasons. A tumour of this kind will be a parovarian cyst burrowing, contrary to rule, into the lower part of the broad ligament. Operative measures are indicated at once, and will probably be successful. But we must remember that the more a thin-walled cyst of this kind burrows, the greater is the chance that it may contain papillomatous growths. Lastly, there is the disadvantage of the capsule, part of which must be left behind.

Let us suppose that we now have to examine a third case. There is a cyst; it is apparently thin-walled, but it is quite tense. The subcutaneous veins in the abdominal parietes appear a little or even markedly dilated, and the patient may complain of a certain amount of debility. The upper part is broad. On the surface of the tumour above one groin is a firm body, broader above than below; sometimes it lies almost centrally, and usually it can be slid about on the cyst to a limited extent. Not rarely the cyst seems bilobed or double. On vaginal examination the cyst is found well engaged in the pelvis. The cervix is much displaced laterally and more or less forwards according to the size of the tumour; on bimanual palpation it is found to be continuous with the firm body above the groin. In other words, the uterus is drawn high up on the anterior surface of the cyst, and frequently a tense cyst can be felt in the vagina on one side of the larger cystic tumour, and may be traced rising by the side of that tumour above the pelvis. The disease in that case is bilateral.

This kind of cyst is very characteristic,—an oval tumour like an ostrich's egg, with the narrow end occupying the pelvis, whilst the uterus lies on its surface. It is known as papillomatous cyst of the broad ligament, and is often bilateral. The removal of such a growth is a matter of some urgency, but at the same time prognosis must be guarded; for there are clearly two forms of papilloma, one malignant and the other apparently a product of inflammation, and the distinction between the two is not always easy, even with the aid of the microscope. The papillomatous masses may be found at the operation diffused widely beyond the cyst walls, over the visceral and parietal peritoneum, a condition usually malignant, yet the diffused growths not rarely disappear entirely after the cyst is removed. Again, the base of a papillomatous cyst is sometimes irremovable because the growths have penetrated its walls and invaded the pelvic cellular tissue; yet even that condition is no proof of malignancy. In one case where I operated I was obliged to leave a mass of papilloma in the pelvis, including a square inch or so of the cyst wall; yet four years later the patient was free from any sign of recurrence, and no deposit could be felt in the pelvis, so the growths must have been of the inflammatory type. On the other hand, a few warts absolutely confined to the inner wall of the cyst may indicate a high degree of malignancy, recurrence taking place within a year or so, even when a pair of tumours have been carefully enucleated. Bilateral disease is so frequent that it is probably safer to remove the opposite appendages even if they appear sound, so as to extirpate the pathological sources of papilloma. Pfannenstiel has shown how common is recurrence in papilloma, either of the ovary or broad ligament; and in Germany he had far greater facilities than we can avail ourselves of for tracing a large series of old cases. I once spared a healthy broad ligament, removing a papillomatous cyst from the opposite side, but had to operate again a few years later. Yet even the second tumour was not malignant, as time has proved. In ovarian papil-

* Exceptionally a large tumour will jamb the uterus against one side of the pelvic wall and prevent it from rising above the brim.

Digitized by Google

loma* such experiences are quite common. Thus this form of broad ligament tumour involves troublesome questions as to prognosis.

As regards diagnosis, we may as well bear in mind that a big lymph cyst in the broad ligament may be indistinguishable from a papilloma. The truth can be alone settled in the patient's interests by an exploratory operation; for if we wait and the doubtful tumour prove to be papillomatous, the consequences will be disastrous, whilst the opening of a lymph cyst is not a dangerous proceeding. It does not recur; it has no true walls—nothing but a boundary of endothelium,—and so cannot be enucleated; and it is best treated by trimming off of much of the distended broad ligament, and drainage of the remainder. We must not be too careless about a "mere pathological curiosity;" we are mistaken if we think it is only of interest to learned young gentlemen who write papers for societies. It is of yet higher interest to the patient. But lymph cysts are rare; I have only operated on three distinct cases,† and in none was diagnosis possible. Of course prognosis is absolutely favourable, since lymph cysts are never malignant.

A true unilocular ovarian cyst occasionally burrows very deeply into the broad ligament, drawing up the uterus on to its front surface. Such a tumour is usually much larger than a papillomatous cyst; the burrowing is a late development. The prognosis is favourable, as the tumour is as a rule innocent; but ovariotomy is relatively difficult in such a case.

Another patient comes under our care. She looks healthy, but says she has been growing big for some time, and thought till recently that the increase in size of the abdomen was really due to fat. We find a very large tumour, solid,‡ elastic, often soft, sometimes very firm. It may even push forward the ensiform cartilage. We feel, if our sense of touch be fairly good and the patient's parietes not too thick, a lump on the surface of the tumour above one groin or over the pubes. On vaginal examination the tumour is found to be considerably engaged on the pelvis. We search for the os and cervix, and often fail to find either. The pelvic part of the tumour comes close to the back of the pubic bones, and the cervix, atrophied and flattened by pressure, may be too high up for detection by the finger. Still the triangular lump above the groin or pubes can usually be defined; and if the cervix can be reached from the vagina, it will be found to be continuous with the lump, which is the uterus flattened out and drawn upon the anterior surface of the solid tumour, just as in papilloma of the broad ligament it is drawn up on the surface of the cyst. It is a most characteristic condition, and signifies that the solid tumour is a fibroid (or fibro-myoma) of the broad ligament, independent of the uterus.

Operation is never urgent in such a case unless there be marked pressure symptoms, but still the patient should be reminded that removal of the tumour is advisable. When the growth is large—which is the rule, as it causes no pain and little inconvenience until it has attained a considerable size and induced the patient to seek medical advice,—its removal involves some risk, as the capsule is very vascular, and a wide chasm in the pelvis is made by enucleation. After a few days, however, perfect recovery may be expected, though inconveniences from ligatures are probable.

Lastly, we may be consulted by a patient who has found out that she has an abdominal tumour. She may be in fair health, but more probably feels as though she were growing weak. There may or may not be occasional hæmorrhage. We detect, without difficulty, a solid mass filling the lower part of the abdomen, or even rising much higher. It is usually elastic and rather soft, but may be hard; its lobulation may be very marked. In short, it bears all the characters of a uterine fibroid. On vaginal examination the tumour is found to be considerably engaged in the pelvis. There may be a short cervix like a nipple running immediately on to the pelvic part of the tumour. Not rarely the os alone can be detected like a circular foramen of small diameter opening directly into the tumour. In a considerable proportion of such cases a deep crescentic fissure is to be felt on the surface of the mass in the pelvis. This fissure is not really in the tumour; if it were so the fissure could only be felt after the lower part of the growth where it lay had sloughed through the uterine mucous membrane and protruded through the os externum. We have not to do, however, with sloughing in this instance. The crescentic fissure is the os externum, extremely deformed through

* Free papilloma of the surface of the broad ligament, which always causes ascites, may be perfectly innocent. Two cases, at least, where I operated over ten years ago, are living and free from any sign of recurrence.

† I have reported one of the three in "Chronic Serous Perimetritis Simulating Cyst of the Ovary and Broad Ligament," 'Journ. of Obstet. and Gynæc. of Brit. Emp.,' vol. i, 1902, Case 4, p. 264.

‡ Exceptionally it may be in part cystic, and more rarely very large fibro-cysts develop in the broad ligament. Diagnosis from papillomatous cyst is often impossible.

Digitized by Google

the invasion by the tumour of one side of the cervix. This crescentic deformity is easy to recognise in clinical examination when its explanation is borne in mind. Lastly, in some cases it may not be possible to define the cervix or the os. In no case can the uterus be discovered by palpation flattened on the anterior surface of the tumour above the pubes or groin, for the uterus is incorporated with the tumour. The sound is a great aid to diagnosis; it will be found to pass into the tumour. But its employment in a case of this kind demands much experience as well as care, and it cannot always be trusted; a soft rubber catheter is sometimes more satisfactory.

This tumour is a uterine fibroid invading the broad ligament. It usually involves the greater part of the cervix, as the relations of the os amply testify; but this is not always the case, and a fibroid of the cervix does not always burrow. This form of morbid growth is dangerous to the patient, and its removal involves considerable risk. The danger of hysterectomy is relatively greater than when a fibroid of the broad ligament is removed, panhysterectomy being specially difficult, whilst hypertrophied uterine tissue makes a bad stump when the uterus is amputated above the cervix. Altogether, however, it is best to remove such a tumour.

There remain two forms of tumour which invade the posterior layer of the broad ligament from behind. Sometimes a huge mass like a fatty tumour is felt in the abdomen, and on vaginal examination part is detected in the pelvis. This is a retro-peritoneal lipoma. Though developed behind the peritoneum, it usually pushes the small intestines aside (to the right, as a rule), so that it will give a dull note on percussion.

Occasionally a soft doughy tumour is felt above the pubes, and below it is found very deeply engaged in the pelvis causing the peritoneum to bulge, whilst the vaginal mucous membrane is everted. The anus is usually patulous as well. On further examination the tumour will be found to be behind the rectum. It is a dermoid of the pelvic connective tissue, entirely retro-peritoneal.

I have endeavoured to make clear the chief features of clinical interest in association with tumours which open up the broad ligament. In any case of a morbid growth of this class we must remember that the tumour should sooner or later be removed, and that, as an inevitable disadvantage, part of a capsule must be left behind. These new growths should be borne in mind, as all who have to do with female patients must understand bimanual examination. For the practice of that method to perfection nothing is more instructive than the definition of the relations of an abdominopelvic tumour of the broad ligaments. Lastly, as I have explained, the ligamentary relations of these tumours play an essential share in prognosis.

THREE CLINICAL LECTURES
ON
HEAD INJURIES.

Delivered at the Medical Graduates' College and Polyclinic.

By T. P. LEGG, F.R.C.S.,
Assistant Surgeon, Royal Free Hospital; Surgical Registrar and Tutor, King's College Hospital.

LECTURE II.

FRACTURES OF THE VAULT AND BASE OF THE SKULL.

GENTLEMEN,—I shall consider to-day injuries to the bones of the cranium under the headings of fractures of the vault and fractures of the base.

Fractures of the vault.—These may be classified into groups:—(1) Those with displacement of the bones—depressed fractures; (2) those without displacement—fissured fractures. And either of these may be simple or compound. The importance of the displacement is solely due to the fact that such important structures as the dura mater, the sinuses, or the brain may be injured. As in fractures in other parts, replacement is the first essential for the successful treatment—meaning thereby restoration of the bone to its normal condition and physiological functions,—so in the case of the skull, replacement or the correction of any deformity is the essential step for the continuance of its normal functions.

Fractures with displacement are due to a concentrated blow, and are localised chiefly to the point of application of the force. The bone is almost always displaced inwards, and the fracture usually involves its whole thickness. In many cases the inner table is damaged to a much greater extent than the outer table, especially in punctured fractures. Occasionally the displacement is outwards, so that the fragments are raised above the level of the surrounding bones. Fractures without displacement are the result of diffuse blows on the head, and the fissure is very likely to extend widely and involve the base of the skull, as well as to be accompanied by intra-cranial complications.

Both varieties are frequently met with at the same time; thus one often sees a depressed area where the main force of the blow was expended, and, radiating from this, fissures running in various

Digitized by Google

directions, involving the base, and accompanied by extensive intra-cranial complications. This case is a typical example :

A young man, æt. 16, fell from a height of twelve or fourteen feet, head foremost. When picked up he was quite unconscious; a scalp wound three inches long just below the occipital protuberance was found. A depressed fracture was easily felt on passing the finger into the wound. He was very restless and almost completely unconscious; there was no bleeding from the ear or nose, or over the mastoid; no paralyses of the face or limb muscles could be detected; his pulse was 88 and quite regular; the left pupil was slightly larger than the right, and both reacted to light. An operation was at once undertaken and a large flap was turned down. An extensive depressed fracture with fissures running in different directions was found. On removing the depressed fragments the dura was seen to be lacerated, brain substance having escaped through the tear. After cutting off all the edges of the skin, which were very bruised, the flap was replaced and sewn up, a drainage-tube being put in. He died the next day. On examining his urine I found that it readily reduced Fehling's solution. *Post-mortem* :—From the gap left by the removal of the fragments, a fissure extended on the right side downwards and outwards across the posterior fossa, ending just behind the groove for the lateral sinus; from the left end of the gap a fissure extended to within 1·5 inch of the external angular process; a third fissure ran towards the vertex from the middle of this one. The posterior end of the right occipital lobe was extensively lacerated.

To the forms assumed by depressed fractures various names have been given, such as gutter fracture, when the depression is long and narrow; pond fracture, when the shape is more or less round; stellate; and comminuted. The most important point is that the inner table is almost always more damaged than the outer when the force is applied from without; if the force is applied in the reverse direction the outer table suffers more. The explanation is that the force causing the fracture becomes more diffused, and acts over a wider area on the table further away from its point of application. At the point struck a certain amount of bending takes place; so long as this is confined within the limits of elasticity a slight flattening of the curve is produced. The particles of bone in the outer table are compressed, whilst those in the inner table are forced apart. In the case of a fracture by a bullet wound the loss of momentum will be an additional factor; the greater the momentum the more cleanly the bullet cuts through.

Punctured fractures.—These form a most important class of cases : first, because they are always compound; secondly, the external wound may be very small, and is liable to be overlooked, especially if it is where the skin is lax and loose, as in the eyelid; thirdly, the dura mater is very likely to be penetrated and the brain damaged; fourthly, the internal table is almost certainly damaged, and its fragments may penetrate the brain or irritate it; fifthly, a foreign body may be present at the bottom of the wound, and unless removed may cause extensive mischief. It is not always easy to localise the foreign body, and even if this can be done by the X-rays it is not always then easy to remove it. There are numerous cases on record where the foreign body has given rise to no trouble, but this can never be foretold in any particular case; and the risks of removal being less than those of leaving it, this should always be attempted.

Fissured fractures.—When these are uncomplicated by a wound they are not easy to diagnose, and are often only diagnosed from the concomitant signs; even if there is an external wound the torn edge of the pericranium may be mistaken for a fissure. In any doubtful case the wound should be enlarged; the cut edges can then be made out and the surface of the bone examined. A fissure would be indicated by the thin red line caused by the blood issuing along its track, and the fact that this is not in the line of any suture.

Fractures of the vault rarely involve one table only; in the region of the frontal sinus the external table may be depressed without any fracture of the posterior wall. The internal table is very rarely broken without the external; sometimes as a result of gun-shot or bullet wound this happens. In children the internal table may be fractured and depressed without the external table being broken, the latter only being bent inwards, forming a sort of green-stick fracture.

The symptoms and signs of fracture of the vault are partly due to the injury to the bone and partly to the effects on the brain. In a non-compound

Digitized by Google

case localised pain, irregularity in the surface, swelling of the scalp, and œdema would be most suggestive; and if to these signs are added symptoms of injury to the brain, the diagnosis becomes almost certain. I have mentioned how a hæmatoma may simulate a depressed fracture. A previous injury, a former operation, or a congenital defect in the skull may cause an error in diagnosis. A man was admitted to the hospital because he had a contused scalp wound over the right parietal eminence. Beneath the wound a very distinct depression in the bone could be felt. After shaving the scalp it was discovered that there was a scar, the result of a previous operation for the removal of a tumour, the gap being caused by the removal of bone. He was intoxicated, which accounted for the restlessness and other symptoms of cerebral injury; he made a complete recovery. Duplay quotes a case of Platner, where the surgeon trephined a man for a supposed depressed fracture due to a fall, the patient being unconscious. It was afterwards discovered that the depression was a congenital defect (Duplay and Reclus, 'Traité de Chirurgie,' tome iii, p. 397). I have quite recently seen a child with a congenital defect in the skull, so large that it admitted the end of the second finger. If this child had been seen after a fall the depression or gap would easily have been mistaken for a fracture.

Evidence of injury to the brain, such as concussion, followed later by compression, is frequently present in cases of fissured fracture. On the other hand, if the depression is slight the symptoms of compression may be quite absent, but later on the patient may become subject to traumatic epilepsy.

In cases of compound depressed fracture the diagnosis is easy; it is evident to both sight and touch, and there may be escape of both cerebro-spinal fluid and brain matter from the wound. The immediate effects on the nervous system are not necessarily marked; the signs of concussion and compression may be entirely absent. But if such cases are not operated on, septic and other effects are certain to follow.

A man, æt. 45 years, fell off a scaffold, and remained unconscious for some time; there was a scalp wound, which was bleeding freely, over the posterior end of the left parietal bone. At the bottom of the wound a depressed fracture was easily felt extending over two inches of the bone. His pulse was 88; there was no paralysis or rigidity of any muscle, and no bleeding from the ears, mouth, or nose; the pupils were equal. The fragments were elevated; the dura was intact and therefore not opened. A complete recovery followed, and he left the hospital three weeks later.

Another man, æt. 28 years, was admitted to the hospital, having been thrown from a horse which he was exercising. He put the horse into the stable, walking about 100 yards, and then came to the hospital. There were two scalp wounds at the posterior end of the vertex, and at the bottom of one there was a depressed fracture. Both the wounds were very much bruised and dirty. Cerebral symptoms were quite absent. Trephining was performed, a fracture 1·5 in. by 1·25 in. was exposed, across which an indent was clearly visible; the inner table was extensively splintered, a slight tear in the dura mater being found. All the edges of the wound were cut away and sutured, the bone not being replaced. Three days later some of the stitches were removed, as the wound was œdematous and red, about a drachm of fluid escaping. The temperature was 101·8°, pulse 98. There were no signs of intra-cranial mischief. Some cellulitis of the scalp followed, and the wound had to be opened up to some extent. An area of bare bone was present at the bottom of it, and the dura was covered by granulations. No necrosis followed, the patient making a good recovery.

These cases illustrate the important points: first, the protection afforded by the dura to the brain; second, that very extensive bone injuries may exist without any lesion of the brain, the main force of the blow having been expended on the bones, which yielded and broke, and thus prevented the violence being transmitted to the brain; third, the necessity of thoroughly disinfecting the wound. In the second case this was not done, but only a mild superficial suppuration followed. It would have been better to put in a drainage-tube for twenty-four hours in this case. The prognosis in all these cases depends on the absence of sepsis, the accompanying intra-cranial lesions or their absence, and injuries in distal parts of the body, which are not infrequent.

The treatment of fractures is not always simple. If the fracture is non-compound, in the absence of signs of intra-cranial lesion, an expectant method is adopted. If a compound fissured fracture is

Digitized by Google

present, the condition of the fissure and the bone around it must be carefully investigated, to see if it is infected by dirt, hair, etc. I here show you a specimen in which the hair can be plainly seen driven into the fissure and fixed there. In all such cases operation is indicated. If any depression exists around the fissure, the same treatment is required, the reasons for such treatment being to avoid septic osteo-myelitis and intra-cranial complications. If there are signs of intra-cranial mischief, operation is always indicated, supposing that other injuries in distal parts are not present and in themselves likely to be fatal. In compound depressed fractures operative interference is demanded at the earliest possible moment. Here the reasons for such treatment are very definite. They are: (1) the element of sepsis must be removed as soon as possible; (2) if the depressed fragments are not elevated or removed, symptoms of intra-cranial mischief are certain to come on sooner or later; (3) an intra-cranial lesion may be present, which can be dealt with at once, such as hæmorrhage, a wound in the dura which can be sewn up, or some lacerated brain substance may require removal; (4) complete exploration of the injured parts.

In cases of simple depressed fractures, one great source of danger is not present. Sepsis being excluded, surgeons formerly advised that operation should not be undertaken in these cases, in the absence of intra-cranial complications. With our present methods of preventing sepsis I think that in any case operation should be performed. It is true that some cases do recover permanently and are followed by no ill consequences, such as traumatic epilepsy; but in a large number of patients such a favourable result is not obtained, and the patient is operated on for one of these complications with a not very hopeful prognosis. This is due to the fact that conditions have followed which cannot be entirely remedied by an operation. Moreover the risk of the operation under these conditions is slight; in 1882 Walsham showed that the mortality of trephining for cases of traumatic epilepsy was 10·6 per cent. At the present time the mortality is between 4 and 7 per cent. Wagner put the mortality for immediate trephining at 1·23 per cent. Therefore I think that even in the absence of evidence of intra-cranial lesion, and as the internal is practically always more affected than the external table, operation is the correct line of procedure. There is perhaps one exception to this rule—the case of children with pond-shaped fractures, unless the depression is extreme, in which case the inner table is likely to be splintered. Owing to the elasticity of the skull bones in children—that is, in young children or infants,—as the brain expands the depression is obliterated. I have seen several cases of saucer-shaped depressions in children who have had no symptoms of injury to the brain, and the patients have got quite well. If there are signs of any intra-cranial mischief, exploration is the proper treatment.

Mr. Jacobson, in his book, 'The Operations of Surgery,' divides these cases of simple depressed fracture into three groups from the point of view of treatment: (1) where the depression extends over a considerable area, and where it is slight in degree (not more than one sixth of an inch), especially if the patient be young and the bones yielding, expectant treatment is no doubt the best; (2) where the depression is limited and defined, where the depressed fragment not only affects a small area, but is turned down angularly or edgeways, operative interference should be resorted to at once, even though no symptoms are present, and whether there is a wound or not, to prevent the onset of dangers, immediate and remote; (3) when in doubt as to the severity of the case the surgeon will do best to explore the fracture, especially in fractures of the frontal and parietal regions, owing to the frequency with which these are followed at a later date by epilepsy. And these rules sum up very well the points to be considered. It is not easy to estimate the amount of depression in all cases, so that I should, in adults at least, if the depression were slight, rather advise operation than adopt the expectant method of treatment.

The method of operating.—The incision in the skin should always be made so as to form a flap, the base or attached edge being downwards, so that its blood-supply may be uninterfered with. This flap includes the whole of the tissues down to the bone, the pericranium being detached with an elevator. The flap should be a large one, to fully expose the site of the fracture, and so that the cicatrix shall not be over the gap in the skull. Sometimes the original wound may be employed in making the flap, after having pared its edges completely. The great advantage that a flap has

Digitized by Google

over a crucial incision is that it diminishes the liability to cerebral hernia.

Treatment of the bone.—Sometimes the fragments are so loose that they can be easily removed by a pair of sequestrum forceps. Any more depressed and fixed pieces should then be raised by an elevator passed between them and the dura. In doing this care must be taken not to damage the dura by tilting the fragments, and if removed they must be drawn out horizontally. Any sharp edges are then cut away by bone forceps. If the fragments are not detached, it is first necessary to remove a crown of bone from the adjacent firm bone, close to the fracture, a sufficiently large circle being removed to enable the forceps or elevator to be passed beneath the depressed portion. If more bone should require removal to satisfactorily expose the parts, this can readily be done by Hoffman's forceps. A large amount may require removal, and any fragments removed should be carefully preserved, with a view to their subsequent replacement.

The dura mater is carefully examined for any wound and any protrusion of brain matter. This is carefully removed and washed with an antiseptic, such as 1 in 2000 perchloride of mercury. The hole in the dura is sutured if quite small; if larger it is also sutured and a drainage-tube put in. The suturing is done to prevent the formation of a hernia cerebri, the drainage-tube providing for the escape of any exudation. Secondly, any bleeding vessel on the dura is ligated. Thirdly, note any bulging of this membrane, indicating increased intra-cranial tension, due possibly to hæmorrhage or effusion of fluid. In such a case the membrane is incised in a curved manner a little distance within the margins of the aperture in the bone, a small incision being made at first, enlarging it if required. After having dealt with the conditions found inside the dura, the incision in it should be sewn up, a small aperture being left, if thought necessary, for a drain. Fourthly, the pulsation and colour; absence of the former may be due to the depressed fragments, or to increased intra-cranial tension from hæmorrhage or serous effusion.

Replacement of the bone.—That the bone will live and fill up the gap if replaced is a well-ascertained fact. The replacement of the fragments is not always possible or advisable. If the amount of bone removed has been small, it is unnecessary to replace it; but if a large area has been taken away and the fracture has been a simple one, it is well to diminish the size of the gap as far as possible by replacing the bone, provided the dura is not depressed. If this is the case the replaced fragments will lie below the level of the rest of the skull and not unite to it in their proper position. In compound fractures it is usually impossible to replace the fragments, owing to the difficulty of cleansing and disinfecting them, without at the same time destroying their vitality. The advantages of replacing the fragments are that no soft spot is left in the skull, and the diminution of the liability to hernia cerebri. To preserve their vitality during the operation, the fragments should be placed in sterilised normal salt solution at a temperature of 100°, or they may be tucked away under the scalp at the margin of the flap. As substitutes for the bone removed a variety of materials have been proposed—thin sterilised platinum or gold foil, or a thin sheet of silver, placed between the dura and internal table, or between the pericranium and external table.

The margins of the flap should be accurately united, the edges of the original incision being pared and sewn up. If drainage is required—and it often is, especially in compound fractures,—the tube should be inserted through a separate opening in the base of the flap. The tube can be removed in twenty-four to thirty-six hours.

In punctured fractures the treatment is similar, the pin of the trephine being applied on the adjacent sound bone, the crown of bone removed including the whole of the punctured portion, especial care being taken to remove the affected parts of the internal table and any foreign body.

Fractures of the base.—Fractures of the base of the skull may be due to direct or indirect violence. In the former the violence is applied directly to the base of the skull, as when the condyle of the lower jaw is driven upwards and backwards against the glenoid fossa, or the vertebral column is forcibly driven on to the occipital condyles. A foreign body may be driven up the nose or through the orbit, producing a fracture of the base in the anterior fossa. The mechanism of these fractures is easily understood.

Indirect fractures are more frequent than the direct ones, and it is of some importance and interest to consider how they are produced.

Digitized by Google

Three chief theories have been held to explain them. The first and oldest was that of *contre-coup* or the vibratory theory. On this theory the vibrations set up by the blow travel in all directions, and are brought to a focus again at the point opposite to that struck. As a result the bone fractures here, radiating or stellate fissures being produced. If the skull were a sphere of uniform shape and thickness, this theory would hold good. It is now generally abandoned in favour of Aran's theory, or the irradiation theory, and the compression or bursting theory. According to Aran, fractures of the base are always due to the direct continuation of a fissure from the point struck, the line of fracture passing by the nearest route to the base. By numerous experiments Aran demonstrated that this occurred in numbers of cases, but in many of his experiments he used a great amount of violence, much more than may cause an ordinary fracture after an accident.

Felizet pointed out that the skull consists of thin portions alternating with thicker portions of buttresses. The latter are arranged in sets. On each side there are two sets, one placed anteriorly, formed by the orbito-sphenoid, and the other, placed posteriorly, by the petro-mastoid. At the front of the skull is the fronto-ethmoid ridge, and at the hind end is the occipital crest. These strong ridges meet in a point about the sella turcica, which has been termed "the centre of resistance." At the base of the brain in that region is the large subarachnoid space. Thus the brain is protected from the jars and blows received in ordinary locomotion due to impact of the vertebral column on the occipital condyles, the vibrations being in part transmitted to the vault through these buttresses, and also through the fluid in the subarachnoid space. On the other hand, blows received on the vertex are carried by the same buttresses to the base and distributed through the fluid, and thus the brain is protected from the full force of the blow. According to Felizet, the line of fracture passes downwards in the thin intervals between the buttresses. Fractures often occur without any fissuring of the vault, and, moreover, it is not uncommon to find the thick parts of the base fractured. A good example, with a drawing of the specimen. is recorded by Mr. Targett in the thirty-ninth volume of the Pathological Society's 'Transactions,' the middle fossa and petrous bone alone being involved. Von Wahl gives an illustration showing an isolated fracture of the base having no radiating fissures in connection with it from the point of application of the force. The clinical history of the case showed that the fracture was due to a blow in the frontal region.

The vault of the skull is highly elastic; this is borne out by both clinical and experimental evidence. The base, owing to its numerous foramina and unequal thickness, is much less elastic and less able to transmit any vibrations propagated to it. Hence the frequency of numerous fissures when the base is broken. On this difference of elasticity is based the compression and bursting theory. When a blow is received on the skull it changes its shape, being compressed in one direction and distended in another. If the alteration in shape exceeds the limit of elasticity, then fracture will take place at the parts subjected to the greatest stress. As the base is the weakest part it is most likely to give way. "Thus transverse compression of the skull gives rise to partial or complete transverse fracture of the base; compression of the skull in its long axis to a partial or complete fracture of the same kind; while if the line of the force is oblique, the line of the fracture is oblique also. Compression of the skull in a vertical direction between the vertex and the vertebral column gives rise to fracture by crushing or compression, which usually takes the form of a circular fracture in the neighbourhood of the occipital foramen" (Tillmans). A very large amount of experimental work has been done on this subject, particularly by the German surgeons. How far the theory can be held to explain the conditions found in fractures of the skull is open to question. In fact, none of the theories is sufficient.

One or all the fossæ of the skull may be involved, the middle being most frequently affected, and not infrequently the petrous bone. These fractures are usually compound, the line of the fissure communicating with the nose, the naso-pharynx, or the middle and external ear. Hence the chief dangers of the injury are septic complications, which are further increased by the difficulty in applying efficient antiseptic applications. In the occipital region, fractures involving the foramen magnum and the inferior occipital fossæ only may be simple.

In children fracture of the base is comparatively

Digitized by Google

rare. In Mr. Battle's list* there were eleven patients under five years of age out of a total number of 168. At the Royal Free Hospital in 1901 there were four post-mortems made on children dying from this injury, the ages being 2, 3, 2, and 10 years respectively. In three of the cases the fissures in the base were extensions from fissures in the vault. These four cases were all admitted during the four months May to August. I have been unable to find any record of any more cases during the last few years at that hospital. The youngest case I have been able to find a record of occurred in a child thirteen months old ('Clin. Soc. Trans.,' vol. xiii, Mr. Eve).

The signs and symptoms of fracture of the base are best considered under the following heads:

1. Those due to injury of the bone.

2. Those due to injury of the dura mater and brain.

3. Those due to injury of the cranial nerves. Owing to the close proximity of these important structures they are frequently affected, and give rise to characteristic signs.

The evidence of fracture of the bones is hæmorrhage, and if this has certain features it is of great importance in making the diagnosis.

The blood may escape from the ear, the nose, the mouth, or naso-pharynx. But to be of diagnostic value the bleeding must be persistent, and either abundant at the onset or by its persistence the total quantity lost must be great.

Hæmorrhage from any of these places is frequently present after injury when there is not a fracture of the base of the skull. The blood may come from a scalp wound and fill up the external auditory meatus. In such a case cleansing the meatus by cotton-wool swabs and making an examination of the meatus by a speculum will show if the blood is really coming from that canal. If the blood is actually coming from the external auditory meatus, this may be due to other causes than a fracture of the base: there may be a laceration of the canal itself, either by a stab or secondarily to a blow on the chin with or without a fracture of the bony canal, the condyle of the jaw having been driven upwards and backwards. In such cases, by cleansing the ear the actual wound may be seen; the membrani tympani will be intact, and hearing will not be interfered with.

Rupture of the membrana tympani.—This lesion necessarily is present in all cases of fracture of the base, accompanied by hæmorrhage from the middle ear, but it may also exist as an isolated injury. It is not always easy to determine this point. As a rule the hæmorrhage in simple laceration of the membrana tympani is slight and not persistent; occasionally it may be abundant. Hearing is preserved, whilst it is often absent in cases of fracture of the base. The following is an example:

Fracture of the lower jaw; ruptured membrana tympani.—F. C—, æt. 40, was subject to epileptic fits, and whilst walking along the street he had a fit. He was brought to the hospital in a dazed condition, with a dirty wound on the chin and blood-stained discharge from the left ear. He had had the fits for twenty-seven years; eight years ago he said he had fractured the base of his skull and made a complete recovery. On examination it was found that there was a compound fracture of the symphysis of the lower jaw. The membrana tympani of the left ear was ruptured; hearing was perfect, and there was no facial palsy or any other sign of fracture of the base or vault of the skull. The bleeding from the ear continued for some time, and was followed by a great deal of pain and some discharge from the tympanic cavity. This ceased before he left the hospital at the end of a month's stay.

Sometimes there exists an isolated fracture of the mastoid process which gives rise to an extravasation of blood into the mastoid cells and antrum, and from there it extends to the middle ear, rupturing the membrana tympani and causing a considerable loss of blood.

Again, the middle ear may be full of blood, the membrana tympani being intact; this can be seen on examining it by a speculum. Hence in all cases of suspected fracture of the base it is of the first importance to examine with a speculum the condition of the external and middle ears, and I think that many cases diagnosed as fracture of the base of the skull because of the hæmorrhage from the external auditory meatus—"bleeding from the ear," as it is called—are really not of this nature at all. I think it well, however, to treat any case in which there is the least doubt as to the condition as if it were the major injury.

* Battle, "Some Points in Connection with Head Injuries," 'Lancet,' vol. ii, 1895.

Digitized by Google

Epistaxis occurs in cases of fracture of the anterior fossa. The blood in these cases may also trickle down the pharynx into the œsophagus and stomach, and be vomited at a later period. One has only to mention that epistaxis is frequently caused by fracture of the nasal bones or laceration of the nasal mucous membrane to indicate the need of a careful local examination to determine the source of the hæmorrhage. Bleeding from the mouth generally means fracture of the posterior fossa through the basi-occipital and basi-sphenoid.

Late hæmorrhage.—We have been discussing so far the bleeding which shows itself immediately after the accident. There are cases, not very infrequent, in which hæmorrhage may not show itself till some time has elapsed after the injury was received. Thus in fracture of the basi-occipital or basi-sphenoid, vomiting of blood may be the only sign of the hæmorrhage, and may not occur for some hours. In such a case the blood, instead of coming through the nose or mouth, passed into the stomach and there accumulated. It will generally be much altered, and of a black or brown treacly character rather than red, unless very abundant. Ecchymoses are often very misleading. They are most commonly seen in occipital and mastoid regions, or subconjunctival. In the two former regions ecchymosis is often the result of direct violence, and then has no diagnostic significance. But if it appears after the lapse of some time—and this may be as late as the third or fourth day,—it becomes of some diagnostic value.

Subconjunctival hæmorrhage is a frequent occurrence when the anterior fossa is involved, and has the following characteristics:—(1) It appears late—perhaps not till two or three days after the injury; (2) it extends from behind forwards, usually at the outer side of the eyeball, spreading into the lower eyelid, the most advanced portion being the narrowest, the posterior limit not being visible; (3) if abundant, the eyeball is proptosed, as the hæmorrhage takes place first into the cellular tissue of the orbit. If the fracture involves the supra-orbital ridge, the blood readily finds its way into the upper lid, and the distinction from "a black eye" is often impossible. In some cases the hæmorrhage appears immediately after the injury. This subconjunctival hæmorrhage differs from a black eye in these points:—(1) The black eye appears immediately after the injury; (2) it involves both upper and lower lids as well as the conjunctiva, and is thus more superficial; (3) the colour of the blood in the conjunctiva is a brighter red. Sometimes pulsation can be felt in the orbit, either because an aneurysmal varix or a traumatic orbital aneurysm has formed.

The source of the hæmorrhage.—In all cases the blood comes from the bone and the dura mater; if abundant, from one or other of the sinuses, the lateral sinus, the inferior and superior petrosal sinuses, or the cavernous sinus, according to the situation of the fissure.

Escape of cerebro-spinal fluid always means that the subdural space has been opened, and renders the diagnosis of fracture of the base certain. It is most frequently found escaping from the ear, but it may be discharged from the nose. In the rare condition called traumatic cephalhydrocele the cerebro-spinal fluid accumulates under the scalp, forming a fluctuating tumour, and as a rule the fluid in the tumour communicates directly with that in the lateral ventricles. This tumour is generally found in young children; it is situated in the parietal or occipital regions, and there may be a considerable gap in the bones due either to absorption or non-development of the bone.

When the cerebro-spinal fluid escapes from the ear, it may at first be blood-stained; later it becomes quite clear, watery, and limpid. It has a specific gravity of about 1005, is alkaline in reaction, and contains a large amount of sodium chloride and only a minute trace of albumen, and a substance—pyrocatechin—which reduces Fehling's solution like grape sugar. The quantity discharged may be quite small or very large; it continues to escape in some cases only a short time, in other cases for many hours or days. If the fluid which is lost from the ear be collected and chemically examined, it is not likely that it will be mistaken for either the liquor Cotunnii, which is derived from the labyrinth, or any inflammatory exudation from the middle ear.

In cases requiring treatment by mercury perchloride and potassium iodide it is usually necessary to continue the administration of the combination with regularity for some time. "Tabloid" Hydrarg. Perchlor. gr. $\frac{1}{32}$ et Potassii Iodidi. grs. $2\frac{1}{2}$, has been recently introduced to provide a trustworthy means of carrying out the treatment when small doses are required.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 583. WEDNESDAY, DECEMBER 30, 1903. Vol. XXIII. No. 11.

CONTENTS.

PAGE

*Two Lectures on Bronchiectasis. By S. H. HABERSHON, M.D., F.R.C.P. LECTURE I 161

*Three Clinical Lectures on Head Injuries. By T. P. LEGG, F.R.C.S. LECTURE III.—Fractures of the Base; Injuries to the Cranial Nerves; Hæmorrhage and Temperature following Head Injuries 166

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

TWO LECTURES

ON

BRONCHIECTASIS.

Delivered at the Hospital for Consumption and Diseases of the Chest, Brompton, October 21st and 28th, 1903.

By S. H. HABERSHON, M.D., F.R.C.P.,

Physician to the Hospital.

LECTURE I.

GENTLEMEN,—My object in these lectures is to present to you as complete a clinical picture of the disease as I am able, and to illustrate by a large group of cases the common physical signs and symptoms of the disease. I may omit some points that you will find in your text-books, but in the twenty cases that I present to you I shall be able to show all the main features of the disease.

Bronchiectasis is a disease characterised by dilatation of bronchial tubes, rarely primary, and usually secondary or associated with other conditions affecting the lungs, pleuræ, or bronchi.

ACUTE.—This is a rare disease, most often described in children or in the aged. It is almost always associated with acute capillary bronchitis or an acute stage of chronic bronchitis. It is known by the name of *acute bronchiolectasis*, and affects the bronchioles or smaller bronchial tubes. It is to be distinguished from the acute complications (inflammatory and septic) which occur in the course of chronic bronchiectasis. It is rarely possible to differentiate it from the disease with which it is associated.

CHRONIC.—This is the subject of these lectures, and includes the majority of cases.

ÆTIOLOGY.—The causes of bronchiectasis are very various, and I shall describe them under three heads:

1. IDIOPATHIC.—A few cases occur in which no previous inflammatory condition has been dis-

Digitized by Google

covered. It is presupposed that a congenital weakness of bronchial tubes is present with a loss of power in the elastic and muscular tissues of the tubes, so that a very slight cause, such as cough or bronchial catarrh, leads them to dilate on the slightest provocation.

2. INFLAMMATORY.—In the histories of a large number of patients some preceding inflammatory attack has occurred. I may tabulate these causes as (*a*) acute and chronic bronchitis; (*b*) pneumonia (acute and chronic); (*c*) pleurisy, with or without effusion; (*d*) inflammatory conditions secondary to other diseases (chronic lung disease due to tubercle or cancer, though compression may bear some share in producing the condition); (*e*) emphysema: this is more often a result than a preceding cause.

3. OBSTRUCTION OF TRACHEA OR LARGE BRONCHI, OR SOME PART OF THE AIR-PASSAGES.—(*a*) Foreign bodies in the air-passages: this is a common and not infrequent cause of bronchiectasis. It leads to dilatation on each side of the obstruction. It may even affect the opposing lung. Such cases are usually rather rapidly progressive. (*b*) Compression by aneurysm, tumours (malignant, hydatid, or congenital), or even from the compression of an abdominal tumour. Destructive necrosis of lung tissue is more often produced, but if the pressure is confined to a main bronchus it is not at all uncommon to find dilatation of bronchial tubes. We have a specimen in our museum of a large hydatid of the right kidney, producing compression and induration of the base of the right lung and a moderate bronchiectasis. (*c*) Stenosis of trachea or bronchi as a result of syphilitic or other ulceration is a rare cause. (*d*) *Adenoid vegetations:* one of the cases that I shall presently show you is that of a child admitted for adenoid vegetations of the naso-pharynx associated with bronchitis. After the operation for the removal of these growths the bronchitis rapidly improved, but the physical signs at the bases, associated with the symptoms and character of sputum and clubbed fingers, led to the diagnosis of bronchiectasis. There is little doubt that, whether directly through obstruction, or indirectly through the influence of the catarrhal conditions of bronchi that the condition incites, adenoid vegetations are the preceding cause of many of the cases of bronchiectasis in children.

IMMEDIATE, INDUCING, OR PHYSICAL CAUSES OF BRONCHIECTASIS.—There are many grounds for believing that dilatation of the bronchial tubes is due to more than a single cause. Much controversy has arisen since the days of Laennec, and many conflicting views have been expressed. There is no doubt that the healthy bronchial tube will not dilate even when subjected to considerable physical strain, but once weakened by inflammatory conditions it is influenced by a variety of causes which tend to cause distension. I will enumerate the various inducing causes that have been suggested.

1. Forced expiratory effort caused by frequent cough. Jenner, in his classical essay on emphysema, has shown that the main effect of cough is to distend the air-passages and alveoli.

2. Dyspnœa with increased inspiratory effort has some influence in increasing pressure and thus distending the bronchial walls.

3. Neither of these causes will produce dilatation of bronchi unless they are associated with loss of elasticity and muscular power by previous inflammatory and other conditions (Stokes and Williams).

4. The accumulation of secretions, combined with the influence of gravitation, bear a share in the process (Laennec).

5. In cases of induration of surrounding lung tissue the contraction that frequently ensues may tend to drag on the bronchial tubes and aid their distension (Corrigan, Rokitansky, and Lebert).

All these causes you will find more elaborately discussed in Wilson Fox's work on 'Diseases of the Lungs and Pleura.'

PHYSICAL SIGNS.—There is no single pathognomonic sign which leads us to make the diagnosis of bronchiectasis. Sometimes the signs, at others the symptoms preponderate, and more commonly it is the combination of the two that determines the diagnosis. I do not propose to present to you any physical signs or symptoms that I cannot illustrate in the remarkable set of cases I am able to show to you. I propose to describe the physical signs under three groups or headings:

1. CASES OF BRONCHIAL DILATATION UNACCOMPANIED BY INDURATION OF LUNG.—The signs are dependent on the situation of the dilated bronchus or bronchi. (*a*) These may be *nil* if the dilated bronchus is deeply seated or masked by emphy-

Digitized by Google

sema. In these cases we depend for diagnosis on the symptoms—the appearance and extent and fœtor of the sputum, and the clubbing of fingers. I shall have occasion to show you two cases in which the physical signs are practically absent, but in which the symptoms unmistakably point to dilatation of bronchi. (*b*) If near the surface, a dilated bronchus produces hollow, tubular, cavernous, or amphoric breathing, and râles of varying size and intensity (to be described later). One feature is that the signs vary at different times. If the saccule is full there will be dulness to percussion, absence of air entry, vocal vibrations or voice resonance. At another time the empty bronchus produces all the signs of cavity (cavernous breathing, with pectoriloquy and increase of vocal vibrations). This is especially the case when the dilatation occurs in the globular or sacculated form of the disease. (*c*) Cases of cylindrical bronchiectasis are often associated with bronchitis. As far as the physical signs are concerned they are more often basic, and the diagnosis, apart from the help derived from symptoms, is largely aided by the peculiar character of the adventitious sounds. Croaking râles (Ewart) are commonly heard—a long-drawn, raucous sound, louder and coarser than friction,—râles which are compared by Fowler to distant friction sounds. These are particularly marked in one case of basic physical signs that I shall show you presently. Other signs, such as post-tussive suction, I illustrate by one case, and Laennec's veiled puff, which is a peculiarity of the inspiratory murmur, which commences at a low pitch and suddenly ends in a manner that is most lucidly expressed by the term puff. One of my cases exhibits this in a marked degree, a second in a less degree.

Post-tussive suction is, I believe, a sound produced by the sudden and involuntary inrush of air into a cavity with firm but elastic walls that is collapsed by the cough, but that expands by virtue of its own elasticity before the succeeding inspiration ensues. We see it commonly in the case of phthisical cavities, but it also occurs, as I hope to demonstrate to you presently, in dilated bronchial tubes. The veiled puff described by Laennec is not confined to the cases of bronchiectasis. I have often heard what I take to be this sign in cases of excavation due to pulmonary tuberculosis, and I have at present under my care a man who exhibits this in a marked degree. It is supposed that the bronchus leading to a saccule or a cavity is plugged by a viscid and almost valvular mass or flake of secretion, and that this suddenly gives way, leading to a rush of air which enters at the end of the inspiratory effort.

2. Cases Secondary to Preceding Lung Disease.—The commonest causes are an unresolved pneumonia or induration of lung consequent to pleurisy or some chronic form of lung disease, emphysema, etc. The physical signs are those of condensation or fibrosis of the lungs, with dulness, weak or bronchial breathing, or of emphysema. The superadded signs of râles and respiration of peculiar character may also be present, and signs of cavitation if the saccule or dilated bronchus approaches the surface. One of my cases presents the signs at the base of the right lung of an unresolved pneumonia which commenced some six months ago. The clubbing of fingers, the excessive muco-purulent and offensive sputum, and the paroxysmal character of the cough determine the diagnosis.

3. Cases in which the Dilatation of Bronchi is followed by Supervening Cirrhosis or Condensation of Lung (chronic pneumonia), or by Emphysema.—(*a*) Consolidation of lung with all its signs is usually present. The physical signs increase as the disease progresses. Respiration varies from weak breathing to tubular, cavernous, or amphoric respiration. Bronchophony or pectoriloquy, if present, varies extremely in different situations. In a large cavity in a tubercular case the character of respiration and voice resonance is more uniform than in the case of bronchiectasis with numerous dilated bronchi at varying distances from the surface. In many cases this variation in the character of the vocal resonance and respiratory murmur in different situations is very striking. Vocal vibrations are diminished if the saccule is full, if the pleura is thickened, or if emphysema is present. (*b*) Emphysema masks the signs completely, unless the bronchial dilatation is near the surface. The character of râles is, however, an aid to diagnosis, apart from the symptoms of the disease. (*c*) Retraction of lung, contraction of chest, and dislocation of the heart are always present if there is much cirrhosis of lung. The dislocation of heart is always horizontal and not obliquely upwards, as in

Digitized by Google

cases of tuberculosis of lungs (Ewart). This is sufficiently obvious, for in the latter class of case the primary disease is more often situated at the apex of the upper lobe, whereas the bronchiectasis cases are usually basic. I shall be able to illustrate this point forcibly in several cases that I shall presently show you.

SYMPTOMS (*a*) may be those of the accompanying inflammatory disease, but as a rule cough, often paroxysmal, excessive sputum with fœtor of expectoration and clubbing of fingers, guide the diagnosis. (*b*) In cylindrical bronchiectasis the symptoms are often those of the associated chronic bronchitis, with the superadded signs of excessive and often fœtid expectoration. (*c*) In sacculated bronchiectasis the symptoms are often most characteristic and determine the diagnosis.

PAROXYSMAL COUGH worse in the morning and excited by change of posture.

EXPECTORATION usually profuse; comes in gushes, often with retching or vomiting, or in a paroxysm like that of whooping-cough. It is usually excessive, purulent or muco-purulent, and offensive. It is out of all proportion to the cough and physical signs. Sometimes a large amount is expectorated at once, from a few ounces to a pint, and in children the attack is often mistaken for vomiting. Fœtor is often observed only in the morning cough or breath; at other times it persists all day long. The sputum, if allowed to stand, usually settles into two or three layers. The lower layer consists of thick yellow or greenish-yellow, almost solid deposit. Above this is a watery layer, and on the surface a frothy and often semi-purulent layer. Microscopically the sediment consists of occasional casts of bronchi, pus-cells, granular detritus—fatty globules and acids and crystals of cholesterin, with bacteria and micro-organisms, leptothrix, and occasionally streptococci and diplococci.

HÆMOPTYSIS is very common, but more often in tubercular cases. It is occasionally fatal, but more often in streaks, clots, or stained sputum. It is due to a general oozing from an intensely congested mucous membrane, or in its larger degree to ruptured aneurysm or ulceration of a large vessel.

DYSPNŒA is usually due to coincident lung complications—emphysema, chronic pneumonia,—or to cardiac dilatation, bronchitis, or even to pneumothorax or empyema.

PAIN occasionally occurs at an early period. It is rarely a prominent symptom (W. Fox).

CLUBBING OF FINGERS is not invariable, but occurs in the large majority of cases. It is sometimes extreme. It is doubtless a nutritive defect, but whether this is produced by deficient aëration of blood, or by some toxic absorption due to the decomposition of the products of secretion, is not determined.

In my next lecture, when I come to deal with the bony affections of bronchiectasis, I shall be able to show you many radiographs of hands and feet of these cases. No bony enlargement exists in the clubbed fingers or toes, and the swelling and tissue change is in all cases superficial.

THE DIFFERENTIAL DIAGNOSIS is not always easy.

I. FROM PULMONARY TUBERCULOSIS.—Bronchial dilatation is often associated with pulmonary tuberculosis. At the same time, in some cases of non-tubercular disease the physical signs are purely apical. The diagnosis then depends upon the absence of tubercle bacilli and on the presence of the typical symptoms of the disease.

The differential diagnosis is obtained from—

1. *Physical Signs.* — (*a*) The variable nature of pectoriloquy and cavernous breathing in different situations thus differing from a large cavity. (*b*) The character of râles—croaking; like distant friction; very large and metallic. (*c*) Superadded sounds, Laennec's veiled puff, post-tussive suction, etc. (*d*) If contracted lung—horizontal dislocation of heart as opposed to contraction obliquely upwards in tuberculous cases.

2. *Symptoms.*—I am able to show you one case of apical signs (extremely slight), and two cases of pulmonary tuberculosis in which the diagnosis is entirely determined by symptoms, and not by physical signs. The former case is free from tubercle bacilli in the sputum (six examinations); and in the two latter cases, though tubercle bacilli have been found, the form and character of the expectoration, with the paroxysmal character of the cough, the fœtor, and the extreme clubbing of fingers, leads to no doubt or uncertainty as to diagnosis.

3. The absence of tubercle bacilli in the sputum affords an important corroboration of the facts observed from symptoms and physical signs.

4. The comparatively healthy appearance and

Digitized by Google

general well-being of the patient in all uncomplicated cases has an important bearing upon the diagnosis and prognosis of the disease.

II. The Discharge of a Localised Empyema through a Bronchus.—In this case (as in that of bronchiectasis), clubbed fingers, purulent and offensive sputum, and pulmonary signs of condensation of lung and of thickened pleura are present. The diagnosis is sometimes one of extreme difficulty. The history often aids the diagnosis.

III. Chronic Fœtid Bronchitis.—Cases have been recorded in which, in the course of a chronic bronchitis, the sputum becomes offensive and accompanied with much fœtor. The evidence of a fœtid bronchorrhœa is undoubted, but there is little doubt that some cases thus diagnosed are due to a dilatation of bronchial tubes.

IV. Gangrene and Abscess of Lung.—True gangrene of lung is ushered in by sudden symptoms of collapse and high temperature, with excessive fœtor of expectoration. The smell is peculiar, and possesses some features that differentiate it from the bronchiectatic odour. The acute onset of gangrene and the fulminating symptoms entirely separate it from the chronic symptoms of bronchiectasis associated with fœtor of expectoration. In the septic complications that occur it is sometimes a matter of great difficulty to decide whether gangrene of lung has supervened.

Abscess of the lung may occur as a sequela of pneumonia, or in consequence of an embolus of a pulmonary vessel, or from an infarct. The symptoms originally acute may become chronic, and lead to a discharge of fœtid pus or muco-pus. The diagnosis is not always easy.

V. Offensive Discharges of Mucus may occur which are derived from the nasal passages (ozæna) or from the pharynx. Pharyngeal mucus can be very offensive in the early morning in cases of chronic catarrh. All these cases must be eliminated in determining the diagnosis.

I now proceed to demonstrate to you some twenty cases of bronchiectasis. I have described some of them to you already, and you will find all the chief physical signs and symptoms illustrated in one or other of the cases. Two cases present comparatively few physical signs, but the symptoms, the character and fœtor of expectoration, the absence of tubercle bacilli, and the clubbing of fingers make the diagnosis certain. In one case there is a large sacculated bronchiectasis near the surface. If the saccule is empty you will note the cavernous breathing and pectoriloquy. If full—and this appears to be the case to-day—you will find dulness, absence of breath-sounds, and loss of vocal vibrations and resonance. I show you two cases consequent on pneumonia. One presents signs of weak breathing, loss of vocal resonance and vibrations at the base, with typical symptoms of bronchiectasis and marked clubbing of fingers.

The second is a case of chronic pneumonia of eight years' duration, with induration of the base of the right lung and dislocation of the heart horizontally to the right. Again the symptoms are typical.

In several cases with preceding bronchial catarrh or broncho-pneumonia, the consolidation of lung has proceeded coincidently with the dilated tubes. All these cases are basic, and in several both bases are affected. You will note all the signs of consolidation of lung with the superadded signs of hollow or cavernous breathing, pectoriloquy varying in different situations, and with râles presenting the characters I have described. In two cases the dislocation of heart is extreme, and you will observe how different the horizontal position of the heart is from the case of the heart when displaced by a contracted pulmonary cavity. The superficial area of cardiac dulness does not in any case extend above the fourth costal cartilage.

One case of chronic pulmonary tuberculosis is shown. The diagnosis in this case rests upon the symptoms. Further, one case of chronic bronchitis with cylindrical bronchiectasis depends for diagnosis partly on symptoms and partly on the peculiar character of the râles.

Finally, you will note in one case the peculiar respiration which changes in pitch towards the close of inspiration, and is a good example of Laennec's veiled puff. One case ought to present the physical sign known as post-tussive suction, while in many of the cases you will observe the croaking and creaking character of râle and the extraordinary jargon of sounds which is so frequently an accompaniment of cases of bronchiectasis.

Digitized by Google

THREE CLINICAL LECTURES

ON

HEAD INJURIES

Delivered at the Medical Graduates' College and Polyclinic.

By T. P. LEGG, F.R.C.S.,

Assistant Surgeon, Royal Free Hospital; Surgical Registrar and Tutor, King's College Hospital.

LECTURE III.

Fracture of the Base; Injuries to the Cranial Nerves; Hæmorrhage and Temperature following Head Injuries.

Gentlemen,—Certain cranial nerves, owing to their anatomical position, are more likely to be affected than others. Thus the seventh and eighth pair are most frequently involved: then the oculo-motor nerves, of which the sixth is most frequently affected. The optic nerves are sometimes damaged, but the remaining cranial nerves are rarely affected.

The symptoms of paralysis of these nerves are so well known that it is unnecessary to describe them in detail. It is necessary, however, to discuss certain features of this paralysis, which may be complete or partial, and which may be evident immediately after the injury or only come on after the lapse of hours, or even days. The following case* illustrates very well these points, viz. late onset of the paralysis and the involvement of several of the cranial nerves, as well as the late appearance of the hæmatoma, of which I spoke in my last lecture. The patient, a man æt. 25, a labourer, was admitted to King's College Hospital on October 3rd, 1900, having been struck by a beam on the left side of the vertex of the head. On admission, he was quite conscious and answered questions readily. He vomited once soon after coming to the hospital. There was a large lacerated wound involving the left upper and lower eyelids. There was no bleeding from the nostril or from either ear; there was no facial palsy, and he was not deaf. The pupils were normal; pulse 46 per minute. For the next two or three days he remained in a drowsy state; at nights he was very restless and slept badly, but was not at all irritable. His pulse remained slow, varying between 52 and 60 per minute. Three days after admission he was found to be deaf in both ears, and on October 9th—six days after admission—he was quite deaf. The right facial nerve was paralysed; he could not close his right eyelid or wrinkle the right half of the forehead to the same extent as the left half. There was no aphasia. Nothing abnormal was detected on examination of the middle ear, but he could not hear a tuning-fork applied to the skull bones. When the bandages were removed on October 11th, a large hæmatoma was found over the mastoid on the right side. On October 12th the external rectus of the right eye was found to be completely paralysed; the third nerve was unaffected. A week later the paralysis of the sixth and seventh was unchanged; he did not seem to be quite so deaf; his pulse was now 72 to 80 per minute; the hæmatoma was being absorbed. On November 3rd the deafness was still improving, but the paralysis of the sixth and seventh nerves was unchanged. The facial muscles, tested electrically, showed commencing reaction of degeneration, and they were atrophied to some extent. When he left the hospital at the beginning of December, nearly two months after the injury, there was some improvement in the condition of the sixth and eighth nerves, but not in that of the seventh nerve.

In this patient there was probably a fissured fracture of the base of the skull and some laceration of the brain leading to hæmorrhage. The hæmatoma over the right mastoid process was probably caused by a fracture of the base, and not by a blow in that region. Its late appearance suggests this, especially as on the left side of the forehead and face, there was a lacerated wound indicating where the blow was received. The sixth, seventh, and eighth nerves were probably pressed on by an intracranial hæmatoma, and the changes in the facial muscles show that the seventh nerve must have been severely damaged, or it may have been involved in the callus. The deafness was possibly due to effusion into the internal ear. He understood and answered questions put to him on paper, and was not aphasic. The sixth and eighth nerves were not as severely affected as the seventh, because they were recovering when he left the hospital.

* 'King's College Hospital Reports,' vol. vii, p. 185. Under care of Mr. Rose.

Digitized by Google

Immediate paralysis of the nerves is caused by their being torn or bruised at the time of the injury being received. The seventh, from its position inside the bony canal of the aqueductus Fallopii and internal auditory meatus, is most likely to be affected in this way; the paralysis is complete and permanent, followed by atrophy of the muscles. Late paralysis of the seventh occurs some time after the injury; in the above case six days after injury; usually the paralysis comes on between the second and eighth day (Demoulin). It may come on gradually, and is due either to the pressure of extravasated blood or to the secondary periostitis which accompanies the repair of the bone. In other cases the late onset is due to an otitis media following the rupture of the membrane, or this disease may have pre-existed, and become worse after the injury. The paralysis may be slight at first, rapidly developing during the succeeding days, and remaining stationary for about a fortnight; then recovery slowly follows, and is as a rule complete in a month. A good prognosis may therefore be given (Demoulin).

The eighth nerve is often affected; this is not always found out, as the patient is unconscious. The terminations of the nerve in the internal ear may be destroyed, more or less deafness of a permanent type resulting.

The oculo-motor paralyses.—The sixth is most frequently affected—the paralysis may be due to a simple contusion without fracture. More often it is due to fracture of the apex of the petrous, which is crossed by the nerve. The paralysis may be immediate or late, and the prognosis depends on the cause. It may recover completely; in other cases the paralysis has remained stationary, or the squint has become more marked by the contraction of the opposing muscle.

The third nerve is occasionally affected: total paralysis is rare; partial paralysis is more common. In the latter group of cases fracture of the roof of the orbit, or compression by hæmorrhage, is the cause. Ptosis is the commonest symptom, and dilatation of the pupil may also be present. I shall presently quote a case in which this nerve was partially paralysed together with the optic nerve.

The optic nerve.—Lesions of this nerve are more frequent than is often stated. After a blow on the head, various visual defects follow in a number of cases; this is an important point to remember, as medico-legal questions may arise in which the ultimate prognosis as to vision is discussed. These defects of vision are due to three sets of conditions: (1) changes in the eyeball itself; (2) changes in and injuries of the optic nerve; (3) involvement of the visual centres. I do not propose to say anything about the first class of cases, except to state that detachment of the retina, rupture of the choroid, hæmorrhage into the vitreous, etc., may all result from blows on the head. The changes which occur in the optic nerve are the result of optic neuritis and atrophy. Lawford and Edmunds* published two papers dealing with the occurrence of neuritis, and concluded that it occurred with much greater frequency in those cases in which the base of the brain or of the skull is involved than in those cases where these lesions are not found, and that this points to basal meningitis as the cause of the neuritis. Battle, in his lectures† on "Some Points in Connection with Head Injuries," published seventeen cases of optic neuritis following head injuries. In four the signs were those of concussion alone. In two out of three fatal cases, at the post-mortem a fracture of the base was found; in the other case, as there was no post-mortem, the diagnosis was not confirmed; the remaining fatal case was due to a bullet wound. In the majority of his cases albumen in the urine was absent, and there was no evidence of syphilis. In two of the fatal cases basal meningitis was present. The date of onset of the neuritis varied, the earliest date noted being the fifth day after the injury, and the latest the twenty-sixth. Mr. Battle thought that the neuritis was more likely to follow in those cases when the posterior part of the skull is affected; there was a fracture of the posterior fossa in nine out of twelve cases in which optic neuritis was observed. Mr. Snell records‡ the case of a youth æt. 19, who was crushed by a cage striking the back of his head, then his shoulders, and then his back. "He saw flashes of light, and then lost consciousness." When he recovered consciousness he was quite blind. Three weeks later there were no decided signs of whitening of the discs. A little more than three months after the accident both optic papillæ were quite white and he was totally blind. Hearing, taste, and smell were

* 'Trans. Ophthal. Soc.,' vols. v and vii, 1885 and 1887.

† 'Lancet,' July 12th, 1895.

‡ 'Trans. Ophthal. Soc.,' vol. xi, 1891, p. 136.

Digitized by Google

quite good. There appears to have been a fracture of the base, implicating the optic canal or the body of the sphenoid, the injury tearing through or damaging the optic nerves or chiasma. And Mr. Snell remarks that "when the injury to the optic nerve has been behind the entrance of the arteria centralis retinæ, the atrophy of the optic discs only makes itself evident after the lapse of some weeks." In vol. xiii, p. 157 of the 'Transactions of the Ophthalmological Society' another case is reported of immediate blindness following a blow on the nasal half of the right lower eyelid; one month after the injury there were signs of optic atrophy in the right disc.

Another patient, a man, received a severe injury to the left side of the forehead, resulting in a fracture of the anterior fossa of the skull. Three weeks later, on examining the fundus of the right eye, the disc was found to be "rather pale, patchy, greyish; cribriform appearance well marked; small vessels seen distinctly." The left disc was uniformly white, and there was total loss of vision in this eye. The visual field of the right eye was measured by the perimeter and found to be much contracted; his vision in this eye somewhat improved at a later date. The lesion was diagnosed to be a laceration of the left optic nerve and chiasma, though it was possible that the symptoms might have been due to pressure of a blood-clot, which must extend back to the chiasma, since the disc of the right eye was affected.*

I have recently had, through the kindness of Mr. Work Dodd, the opportunity of examining a man who was shot at Paardeburg in 1900. The bullet entered just above the middle of the right zygomatic process, and emerged immediately above and below the left external angular process. For three quarters of an hour after the injury he was able to see well. The wounds were dressed and a bandage applied. On removing the latter at the end of twenty-four hours he was quite blind. He never lost consciousness; he was not even stunned, and there were no other symptoms of injury to the brain or base of the skull. On examining the right eye there was paresis of the levator palpebræ, the superior rectus, and the eyeball was on a lower level than the left, and could not be raised so well. The fourth and sixth nerves were normal; the pupil was dilated and inactive. The disc was atrophied, as was also the choroid, and he had no perception of light. The lens of the left eye was opaque; the condition of the fundus could not be made out, but he had no perception of light. As far as I know there was no syphilitic history, and as the patient's vision was quite good till he was shot we must ascribe the atrophy to the bullet wound.

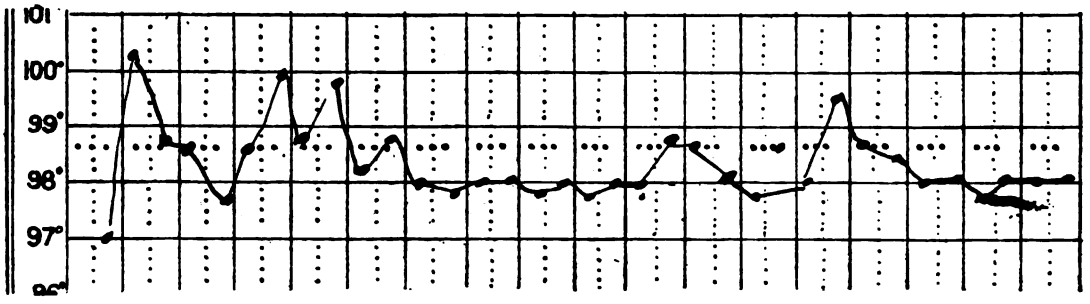


CHART A.—Compound comminuted fracture, left parietal region; trephined; no symptoms except concussion; recovered.

It is evident, therefore, that either optic neuritis or atrophy may follow a head injury. By some, the neuritis is considered to be secondary to a localised meningitis; others think that the inflammatory process starts in the contused brain substance and spreads along the cerebral membranes to the optic nerves. With its onset there is not necessarily any rise in temperature. In many cases there is a fracture of the roof of the orbit, with crushing of the nerve and hæmorrhage into its sheath. Optic neuritis with hæmorrhage around the papilla has been observed after head injuries.* But in those cases where the roof of the orbit and the optic foramen are involved in the fracture,

* 'St. Bart.'s Hospital Reports,' vol. xxxiv, p. 257.

* See also a recent paper in 'Brit. Med. Journ.,' Feb. 21st, 1903, p. 429.

Digitized by Google

primary atrophy of the disc is more frequent. If the injury to the nerve is situated behind the entrance of the central artery of the retina, the atrophy may only make itself evident some weeks after the injury, as in the first and third of the above cases. But if the optic nerve has been injured so far forward that the central artery of the retina is divided at the same time as the nerve, characteristic ophthalmoscopic signs can be made out at once; the arteries of the papilla and retina are bloodless, and the retina soon becomes clouded. These cases of blindness are incurable.

A punctured wound in the orbital region may also lead to loss of vision, the foreign body having divided the nerve. It is important to remember that the external wound may be very slight, that the foreign body may be retained in the orbital fat, where its presence is not always easily detected.

ness in the left half of each visual field. About five months later he had almost completely recovered from the hemianopia. In the 'Transactions of the Ophthalmological Society,' vol. xxi,* there are four cases of injuries by bullets in which hemianopia followed an injury in the occipital region. In all of them the patient was blind immediately after the accident, or was found to be so on recovering consciousness. In one patient, the sight was noticed to be returning on the fifteenth day; in another on the sixth or seventh day; in a third patient, after being blind for two days, sight gradually returned on the fourth day, and on the eleventh day the man had perception of light. In one patient the lower half of each visual field was lost, and subsequently vision was almost completely recovered. In this patient the bullet probably damaged both occipital lobes, but total de-

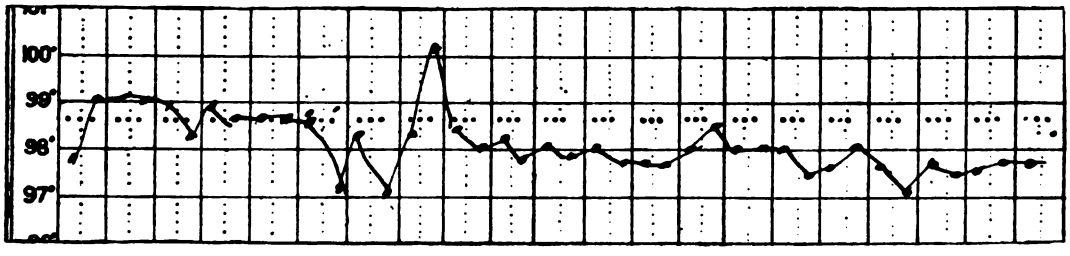


CHART B.—Depressed fracture; trephined; recovered; no signs of injury to brain.

Careful probing of the wound should therefore be carried out.

It will be convenient at this point to consider the other visual affections which may follow head injuries, viz. those following on injury to the visual centres.

A man was admitted to hospital with a compound depressed fracture in the left occipital region. Immediately after the injury his sight was not examined, but four months later he was found to have partial right hemianopia involving almost the whole lower quadrant of the fields of vision. There were no changes in the fundus. Two months later there was considerable improvement in the right eye.* A similar case is recorded by Mr. Page,† the patient being a boy æt. 15 years, who had a compound comminuted fracture in the right occipital region, the brain being also lacerated. On examining the optic discs they were found to be normal, but there was practically complete blind-

struction could not have occurred, as recovery ensued. In two of the cases the lower half of each visual field was lost, and in the fourth the right half of the visual field was absent, the left occipital lobe being damaged. In two of these cases it is definitely stated that the fundi and pupils were normal.

When the occipital lobe is involved, such lesions as hemianæsthesia and hemiplegia may occur. If the left occipital lobe be implicated, certain forms of aphasia may also be detected.

A certain number of the patients recover—some completely, others incompletely. The prognosis is certainly more hopeful than in those cases in which the optic nerve is damaged. Experimentally, the greater portion of both occipital lobes may be removed without causing more than transient visual disturbances, and without causing permanent blindness in any part of the visual field. Schäfer's experiments show that, in order to cause complete loss of vision, not only must the occipital

* 'St. Bart's Hospital Reports,' vol. xxxvii, p. 185.

† 'Lancet,' vol. i, 1901, p. 157.

* Anderson Critchett and Fisher, p. 23, et seq.

Digitized by Google

lobes as such be cut off, but the lesion must extend well forward into the occipito-temporal convolutions.*

The olfactory nerve is sometimes damaged in cases of fracture of the anterior fossa or by punctured fractures involving the cribriform plate. Loss of smell may be the result.

Escape of brain matter occasionally occurs in fracture of the base and makes the diagnosis certain. A fatal termination is the usual result. Escape of brain matter is much more frequent in cases of compound fracture of the vault, and is not necessarily fatal.

From this discussion of the signs and symptoms it will be observed that the diagnosis of fracture of the base is not infrequently very difficult, and may be impossible. There may be an entire absence of symptoms of injury to the bones, or they may only appear after an interval of time has elapsed.

The prognosis of fracture of the base is more favourable than formerly. It is grave both from the point of view of immediate recovery, and the remote complications which may follow. The gravity of the immediate prognosis depends on (1) the co-existence of extensive cerebral injuries; (2) the fact that the fracture is compound, the wound being in situations where disinfection and the prevention of access of micro-organisms is impossible or very difficult. The patient may recover from the immediate effects of the injury only to succumb to some septic disorder such as septic meningitis or encephalitis; (3) on intra-cranial hæmorrhage which, though capable of being successfully dealt with, is accompanied by other cerebral lesions fatal in themselves. If the patient escapes all these dangers, marked changes in the habits and mental characteristics of the patient may follow and persist to the end of life.

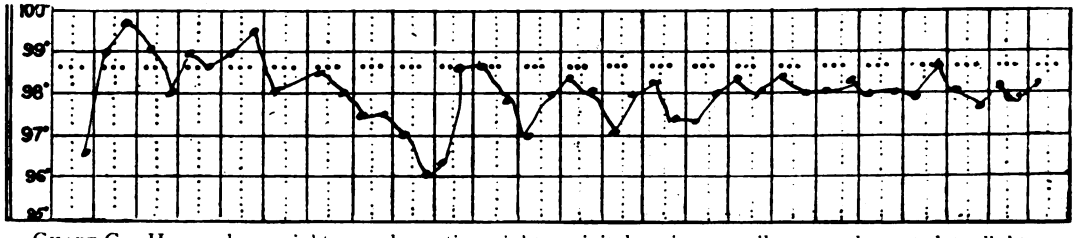


CHART C.—Hæmorrhage, right ear; laceration, right occipital region; pupils unequal, reacted to light; insanity followed; recovered.

It is by attaching to each sign its relative importance that a diagnosis can be made in the absence of such characteristic symptoms as the escape of cerebro-spinal fluid or of brain matter, which are comparatively rare, and which only occur in the most severe cases. From the region in which the signs appear one is enabled to say which part of the base is affected. The presence of a wound in the scalp or of a hæmatoma is of considerable importance in this respect. Or it may be without any of these external signs, there are present the signs of injury to the brain or nerves. These may be evident immediately after the injury was received, or they may come on at a much later period—some hours, or even days, perhaps weeks afterwards. The presence of these symptoms immediately after the injury enables a probable diagnosis to be made, and at a more remote period to always consider the connection between the history of an injury and an apparently spontaneous affection.

* Allbutt's 'System of Medicine,' vol. vii, p. 315.

The following cases illustrate very well the mental changes, sometimes amounting to actual insanity, which follow a fracture of the base.

A man, æt. 46, was admitted to the Royal Free Hospital under the care of Mr. James Berry, having fallen off his cab on to his head. A few months previously he appears to have had a similar fall, and was in another hospital for six weeks. From this injury he appears to have made a complete recovery. When admitted he was quite conscious and able to walk. There was hæmorrhage from the right ear and a hæmatoma in the right occipital region. The pulse was 60. The pupils were unequal in size—sometimes one and sometimes the other being the larger; both reacted to light and accommodation. Next day the pulse was 70; he was quite sensible, and could remember what had happened up to the time of his fall, but nothing of it. Two days later he was drowsy, and on the night of the fourth day after the accident he did not sleep, and got out of bed

Digitized by Google

three times; he became very excitable and restless. On the ninth day after the accident he was talking and chattering incessantly; he had to be constantly restrained owing to his restlessness, and he had a fixed delusion that he was driving his cab. No improvement following, he was sent to the infirmary, as he was quite insane. He has since apparently completely recovered. There was a marked alcoholic element in this case.

In the next case which, by the kindness of my colleague, Mr. James Berry, I have had the opportunity of frequently examining, the mental changes took the form of loss of memory for both recent and past events, and delusions—imagining people were present who were not, talking nonsense, and not recognising those who were in constant attendance on her and whom she had previously known. There is a doubtful history of alcoholism. Previous to the accident the patient appears to have been a bright and cheerful woman, and well educated for her station in life. She was fifty years old, and appears to have fallen down a flight of steps on the evening of February 9th last. She was unconscious when picked up, and was bleeding from the nose and right ear. Vomiting frequently occurred, the vomit containing some blood. There was no bruise on the head and no evidence of facial palsy or any of the limb muscles. The legs were rigid, and she had incontinence. The temperature rapidly rose to 105°. On the 11th she became very irritable, spitting out of her mouth all her food, etc. On the 12th there were signs of right facial paralysis followed by twitching of the muscles on that side of the face. Slight hemiplegia of the right side was also noticed. Then some aphasia developed. She mispronounced words, was unable to read, and could not say where she lived, any attempt to do so resulting in an entirely wrong word being used. At the end of a month there was very little improvement in her condition, and one day she got out of bed "to talk to some of her neighbours and see what they were doing." She was ultimately sent to the infirmary. There was in this patient undoubtedly some gross lesion of the brain of the left side in the Rolandic area and Broca's convolution.

Repair of fractures of the base.—Fractures of the base are repaired by bony callus. This has been denied, but von Bergmann showed in twenty-five cases, by post-mortem examination, that there was real bony union. A long time is required for its formation; it is often imperfectly formed, partly on account of the condition of nutrition of the bone, which is unfavourable (Koenig), and partly by the interposition of substances between the fragments (Duplay). In fractures of the vault bony callus is also formed, but never in excess. It is produced essentially by the diploë, the periosteum playing only a secondary part. When a part of the skull has been removed there is imperfect bony union, a dense membranous cicatrix with an irregular bony periphery filling up the gap.

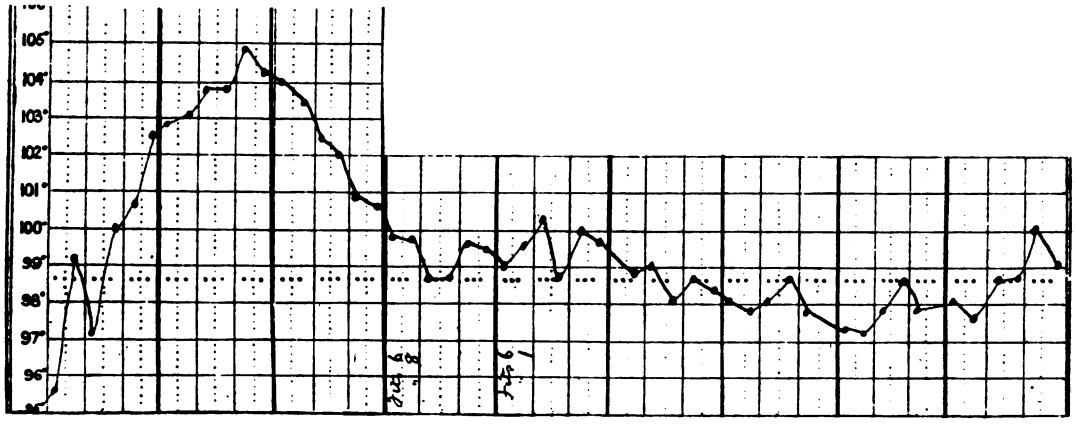


CHART D.—Fractured base; slight facial palsy and hemiplegia; recovered.

Treatment of fracture of the base.—This is essentially symptomatic in uncomplicated cases. Absolute rest in bed in a darkened room, a fluid diet, and regulation of the bowels will meet the indications in such cases. As regards the purga-

Digitized by Google

tive, calomel is the drug which finds most favour. In my experience it is not the most satisfactory or useful.* I find in hospital patients particularly, even after a large dose—say 10 grains,—that no evacuation results, whether it be given in divided doses or in one dose. The administration of a saline, sulphate of magnesia and senna, I have found to be more efficacious, if the patient can swallow. If the patient is unable to swallow, one or two minims of croton oil, mixed with a little sugar or butter, is the best aperient. Both this drug and the calomel should be placed far back on the tongue if the patient is unconscious. To combat the shock, warmth applied by hot blankets and hot bottles (the latter covered with flannel and not placed in contact with the patient) is often sufficient. Stimulants, such as strychnine and brandy given hypodermically, should not be given unless the collapse is very great. It is not necessary, unless for the purpose of cleansing and disinfecting a wound, to shave the head. If the hair is very thick it may be cut short. An ice-bag or Leiter's coils may be applied to the head. It is, however, difficult to keep the bag applied, and if the patient is at all restless the bag is usually on the pillow. Moreover some patients object strongly to it, and I am not sure that in any case the bag has any therapeutic effect.

It is well to syringe out the ears carefully, not using any force in directing the stream of lotion, such as boracic or 1 in 2000 perchloride, and allowing the return stream to escape freely. The nose may be similarly washed out. The ear having been thus cleansed, iodoform may be powdered in, and a small plug of antiseptic wool placed in the external meatus, changing it as often as necessary. Similarly, iodoform may be insufflated into the nostrils. The important point is not to allow any blood-clots to accumulate.

As regards the actual fracture of the base itself nothing can be done. If there are signs of increasing intra-cranial pressure—and this is usually due to hæmorrhage,—this will require treatment in the manner detailed under "middle meningeal hæmorrhage."

An important point in the treatment is, "How soon shall the patient be allowed to get up?" No definite rules can be laid down on this point; each case must be judged on its own merits. The pulse-rate should have been allowed to return to the normal for some days, and should not vary when the patient sits up in bed. Giddiness is another important contra-indication to allowing the patient to get up. Often, so long as the recumbent position is maintained, the patient feels quite well. As soon as he sits up or attempts to get out of bed, marked giddiness occurs. In the presence of this symptom the recumbent position must be maintained; if, however, it is only transient, on assuming the upright position in bed, he may be allowed to sit up, and in a few days to leave his bed. Headache is another contra-indication to allowing the patient to leave his bed, especially headache on slight exertion, such as reading or talking. Drugs may be required to relieve this symptom, the most useful being small doses of potassium bromide and iodide combined with a bitter tonic.

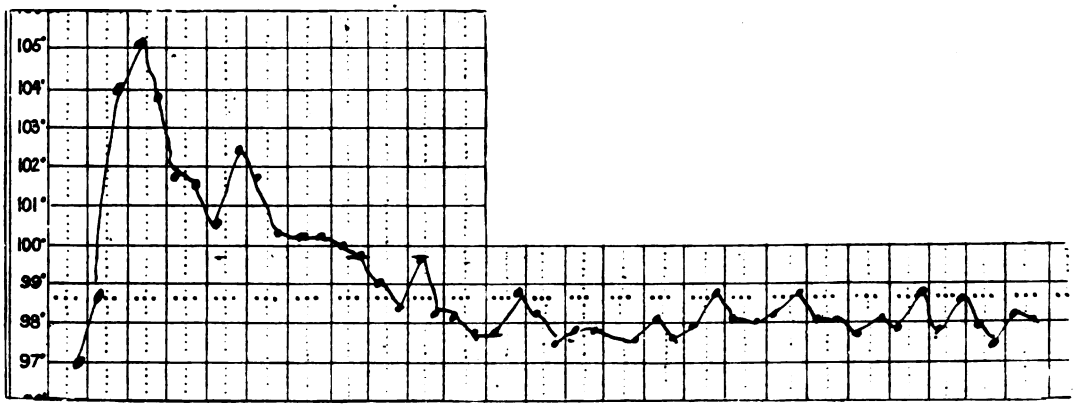


CHART E.—Lacerated brain; many fits; trephined; two ounces of serous fluid evacuated from subdural space; recovered.

* Different specimens of calomel vary much in their efficacy.

Digitized by Google

If these symptoms are absent it is safe to allow the patient to get up and gradually resume his usual mode of life. It is better to err on the side of excessive caution rather than in the opposite direction. And the same rules apply to patients who have had a fracture of the vault or other head injury.

Intra-cranial complications of fracture of the skull may be classified under the headings of—

Hæmorrhage is the most frequent and fatal intra-cranial complication of a head injury. It is found in various positions, and not infrequently in more than one situation in the same case. Thus it occurs between the dura mater and the bone—a common site,—and the blood is then derived from (1) the vessels of the diploë; (2) from the meningeal arteries and veins, especially the middle and, most frequently, its anterior division; (3) one of the

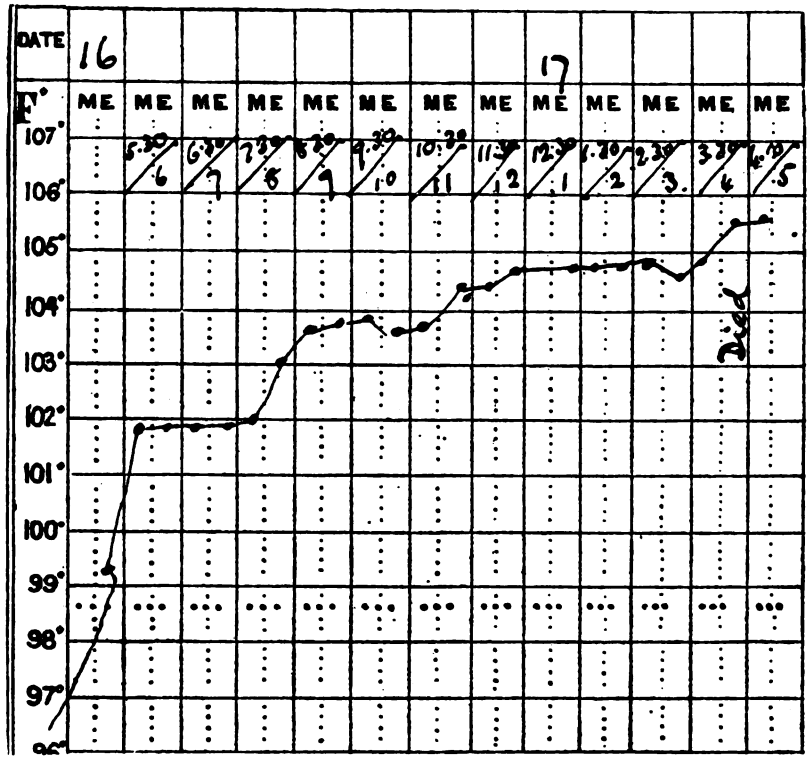


CHART F.—Extra-dural hæmorrhage; fracture of base; lacerated brain; trephined; death in twenty-four hours.

1. Hæmorrhage.
2. Cerebral conditions—
   (*a*) Concussion.
   (*b*) Cerebral irritation.
   (*c*) Compression.
3. Encephalitis—
   Localised.
   Diffused.
4. Meningitis—
   Localised.
   Diffused.

It is impossible to deal with all these conditions in detail, and I propose to speak first of hæmorrhage.

large sinuses, the cavernous, superior longitudinal, or lateral sinus—the latter being most frequently affected. When the symptoms of compression are due to hæmorrhage from rupture of one of these vessels alone, or with such an injury to the other intra-cranial contents that the symptoms of these other injuries are delayed, a constant sequel of events is observed. The patient is first stunned or concussed by the blow; then consciousness is recovered, and the length of time the conscious interval lasts, varies. In some cases it is quite short; in other cases it is much longer and may be hours, the coma coming on gradually and deepening as the pressure of blood increases The ex-

Digitized by Google

planation of this phenomenon is that the dura being adherent to the skull, and especially so at the base, it has to be separated by a considerable degree of force, and hence will offer resistance to the blood being extravasated. Here also is the explanation of why these extra-dural hæmorrhages are localised, and when large, produce a depression on

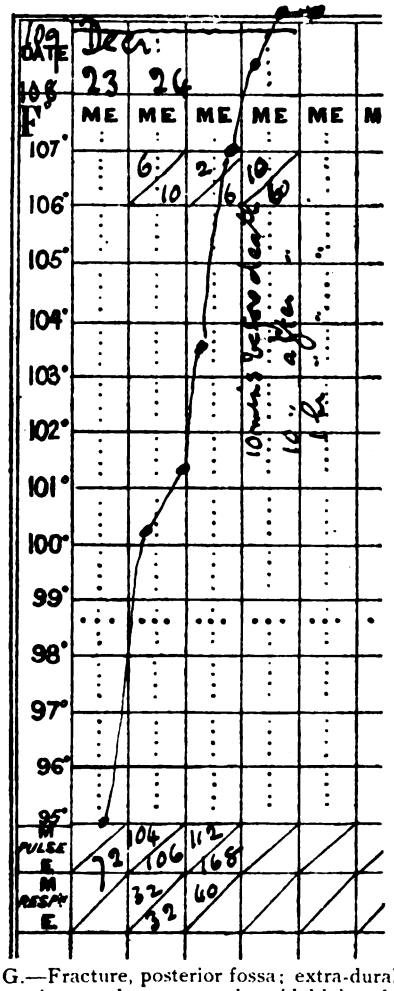


CHART G.—Fracture, posterior fossa; extra-dural hæmorrhage; lacerated temporo-sphenoidal lobe; death.

the surface of the brain. At first, then, if only the meningeal artery or one of the sinuses is torn, consciousness is not lost till the intra-cranial pressure has been greatly increased. And as this increase can only take place gradually the loss of consciousness is gradual, depending on the rate the blood is extravasated. In the case of one of the sinuses being torn, the blood-pressure being low in them, the interval may be very long—in one case at the end of twenty-four hours consciousness was not completely lost, and the patient was only drowsy. If the middle meningeal is torn the interval may be only two or three hours or less. If there are other severe cerebral lesions this train of events may be wanting, and if there is an alcoholic element present the conscious interval may be entirely absent.

Through the fissure in the bones of the vault the blood may pass and rapidly accumulate under the aponeurosis of the scalp, forming a distinct tumour. The pupil on the side the hæmorrhage is taking place may be contracted, whilst the other is normal. Then dilatation may follow, so that frequently the pupils are unequal in size as well as inactive to light. If the cortical motor centres are irritated, spasmodic convulsions, beginning in a definite group of muscles and following a definite sequence, may occur. The pulse at first is slow, often very slow, and as the pressure increases so does its frequency. Besides being slow, the pulse is full and regular at the onset, and then becomes weaker and weaker, and more and more irregular. Such is the clinical history of a typical case of extra-dural hæmorrhage, and it is the form which offers the greatest prospect of successful treatment by surgical means; but on account of the associated lesions a fatal result is very frequent.

Whenever such hæmorrhage is diagnosed or suspected, early trephining must be the treatment. In many cases there is the presence of a hæmatoma or a depressed fracture to indicate where this should be performed. In the absence of such indications, the area of the middle meningeal artery on the side opposite to the affected limb should be exposed by turning down a large flap and applying the pin of the trephine an inch and a half behind the external angular process of the orbit and an inch and a half above the zygoma. (If a fissure is seen on exposing the bone, the pin of the trephine will be applied close to the line of this.) This point is chosen because it is that at which the anterior branch crosses the inferior angle of the parietal bone in the canal which is often found here. Having removed the circle of bone and enlarged the opening thus formed, if necessary, by means of Hoffman's forceps, the clot is exposed and removed. It may be pulsating, which means that hæmorrhage is still going on. The clot is gently detached by means of the finger, a Volkmann's spoon, or other similar instrument, the last portions being removed

Digitized by Google

by irrigation with sterilised saline solution under low pressure. The bleeding may now cease, or it may continue welling up from the base of the skull. If this is the case the hole in the bone must be enlarged downwards and an attempt made to find the bleeding spot, which may be the middle meningeal, as it enters the cranial cavity through the foramen spinosum, or one of the sinuses. The outer surface of the dura should always be examined, as the torn artery will be frequently found lying on its surface. Having found the bleeding vessel, it may be possible to ligate it, or, failing this, to under-run the vessel with a fine suture by passing the silk or catgut through the dura and then tying on the surface over the vessel. If the torn ends have retracted into the canal in which the vessel lies, the walls of the canal may be crushed by a pair of forceps. In many cases plugging by gauze is the most efficient means of arresting the hæmorrhage. In applying the gauze it must not be packed too tightly, as the symptoms of compression may be then kept up or come on as the blood is absorbed by it. It is only very rarely that ligation of the external or common carotid is required in cases of hæmorrhage from the middle meningeal artery.* The wound is closed, a small drainage-tube being inserted if necessary, or if gauze plugging is used the ends are brought out of the wound, and it may be removed in twenty-four to thirty-six hours, either gradually or at once, according to circumstances.

The posterior division of the middle meningeal division may be exposed by trephining at the point where a line from the external angular process drawn backwards to the occipital protuberance crosses one drawn vertically upwards at the posterior margin of the mastoid.

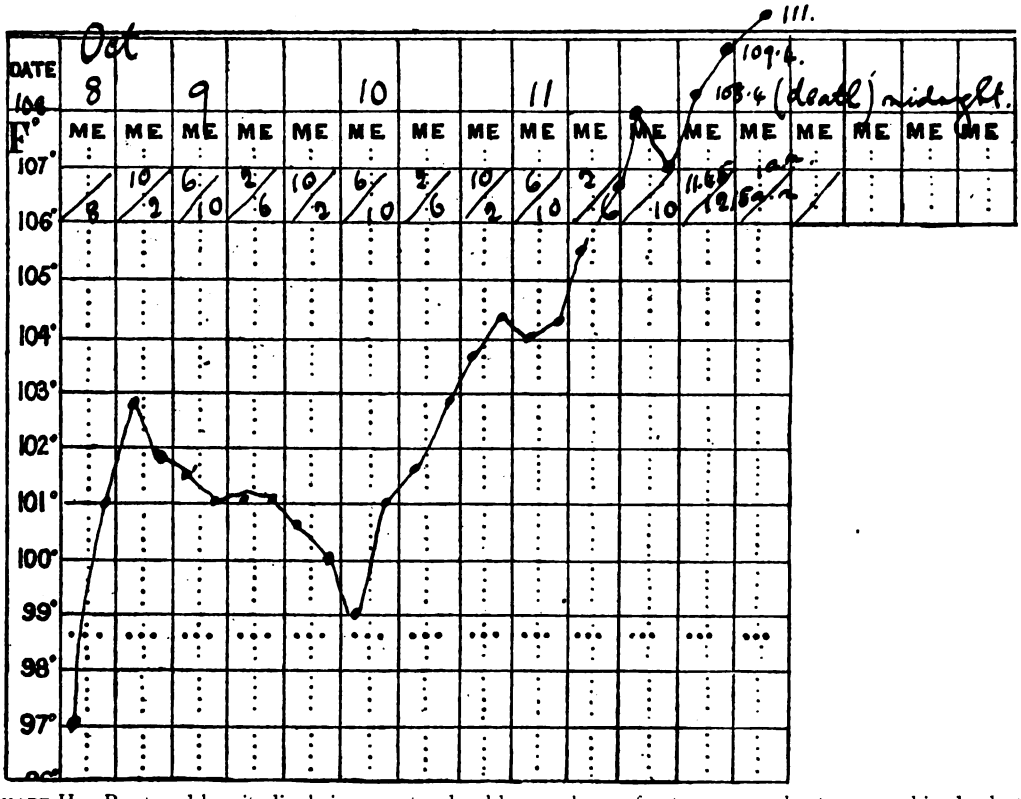


CHART H.—Ruptured longitudinal sinus; extra-dural hæmorrhage; fracture, coronal suture; trephined; death.

Subdural hæmorrhage is caused by (1) rupture of the dura, the blood passing inwards through the rent from one of the meningeal vessels; (2) rupture of one of the sinuses of the dura mater; (3) rupture of the vessels of the pia mater and laceration of the brain. These cases are much more difficult to deal with surgically. If one of the

* Shepherd, 'Brit. Med. Journ.,' vol. i, 1896, p. 905.

Digitized by Google

sinuses is wounded it may be possible to arrest the hæmorrhage by plugging with gauze, by ligature (difficult), or by suturing the edges of the rent. If an extra-dural hæmorrhage is not present, and the dura bulges into the wound and does not pulsate, it should be opened by a curved incision parallel to and a little distance inside the margin of the aperture in the bone. The clot is removed partly by a spoon, and partly by gentle irrigation with sterilised saline solution. Any bleeding points may be gently pressed by a sponge or swab wrung out of hot saline solution. The dura is then sewn up by fine silk or catgut sutures, a small drain being inserted if necessary. Instead of blood a collection of serous fluid may be evacuated from the subdural space, as in the following case:—A man, æt. 42, was admitted to the hospital, having fallen on to the back of his head. He lost consciousness, but soon recovered and walked to the hospital. He seemed to be somewhat dazed, and a bruise extending some distance forwards was found behind the left inferior angle of the parietal bone. Half an hour after admission he vomited, and an hour and a half later he had a fit, which began at the right angle of the mouth; then the right arm, right leg, left arm and leg were affected. He became comatose; the left middle meningeal artery was exposed and found to be intact. The dura did not pulsate. It was incised, and two ounces of serous fluid were evacuated. During the twenty-four hours following the operation he remained in a comatose condition, and had several fits similar to those previous to the operation. An uninterrupted recovery then followed.

The temperature after head injuries.—I wish now to direct your attention to these temperature charts. Chart A is that of a girl who had a compound fracture of the left parietal bone, and who was trephined, a large area of bone requiring removal. She made an uneventful recovery. There were no symptoms pointing to gross lesion of the brain. Chart B is that of another patient who recovered after trephining without any symptoms of injury to the brain. Chart C is that of a man who had all the signs of a fractured base without intra-cranial lesions. Contrast these charts with D and E. In both these patients there were signs of gross lesion to the brain: the patient to whom Chart D belongs had aphasia, slight hemiplegia, rigidity of the limbs; the patient to whom Chart E belongs was trephined, and serous fluid evacuated from under the dura mater (*vide supra*). In both these cases a rapid rise, followed by a rapid fall to normal, is the most striking feature. Both patients recovered. The charts F, G, and H show the rapid rise, and often to a great height, in cases where very severe intra-cranial lesions are present. Each of these patients died. The patient from whom Chart F was obtained had a lacerated cavernous sinus, an extensive extra-dural hæmorrhage, and laceration of the left frontal and temporo-sphenoidal lobes. The base was also fractured. Chart G was obtained from a man who had a fracture of the posterior fossa of the skull, a large extra-dural hæmorrhage, and extensive laceration of the temporo-sphenoidal, frontal, and parietal lobes of the brain. Chart H is the record of a case of a woman who had a rupture of the superior longitudinal sinus, and a large subdural hæmorrhage. There was no laceration of the brain. In the anterior part of the pons there was a minute hæmorrhage.

These charts are typical, and I think one may draw the following conclusions from a study of them and others of a similar kind:—(1) That there may be no rise in temperature, even when the fracture is compound and extensive, care being taken to exclude sepsis. (2) If there is no injury to the brain of a gross nature, the temperature remains normal or nearly so. When there is a slight laceration of the brain the temperature will rise to a slight degree (100°—101°). (3) A rapid rise in temperature, and not infrequently to a great height, always means a severe intra-cranial complication, *e. g.* lacerated brain, hæmorrhage. (4) The temperature chart is of considerable value in prognosis—a continued rise means a fatal result. In those patients who are going to recover the rise is followed by an equally rapid fall (Charts D and E).

The Use of Adrenalin in Anæsthetic Injections.—Braun considers adrenalin of great value as an addition to local anæsthetic mixtures, but finds caution necessary in regard to two points, *viz.*, toxic effects and secondary hæmorrhage. Eight centigrams of cocaine, together with eight drops of one to one-thousand adrenalin chloride solution, have caused death, and even three drops are reported by Enderlew to have produced uniformly untoward symptoms. The author has often exceeded this dosage, however, without bad effects.—*Medical Record*, December 12th, 1903.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. [illegible] CREASY.

No. 584. WEDNESDAY, JANUARY 6, 1904. Vol. XXIII. No. 12.

CONTENTS.

PAGE

*Two Lectures on Piles, their Nature and Treatment. By J. JACKSON CLARKE, M.D., F.R.C.S. LECTURE I ... 177

*Two Lectures on Bronchiectasis. By S. H. HABERSHON, M.D., F.R.C.P. LECTURE II 183

*A Clinical Demonstration at the Medical Graduates' College and Polyclinic. By HAROLD L. BARNARD, M.S., F.R.C.S. 188

** Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

TWO LECTURES

ON

PILES, THEIR NATURE AND TREATMENT.*

J. JACKSON CLARKE, M.B.Lond., F.R.C.S.,

Senior Surgeon to Out-patients at the North-West London Hospital; Surgeon to Out-patients at the City Orthopædic Hospital.

LECTURE I.

GENTLEMEN,—When a patient complains that he is suffering from piles we know that his trouble may be almost any affection of the lower part of the rectum, or of the skin at the margin of the anus; it may be that he has merely a local eczema, or he may be suffering from so grave a disease as cancer. Hence the need of careful clinical examination in every case. Seeing that our patients have so vague an idea of the meaning of the term "pile," it behoves us to be very careful to know exactly the various conditions to which this term is applied in medicine. A list of the chief conditions to which the term pile is applied will best serve to introduce the subject. There are:

1. The cutaneous pile, a small tag of skin at the margin of the anus, commonly caused by an organised thrombus in that place.

2. The external pile, which is a small lump caused by a varicose condition of one or more of the veins at the anal margin.

3. The internal pile, which is composed of mucous membrane and submucous tissue, and of which there are three varieties: (*a*) the *venous pile*, the bulk of which is made up of varicose veins; (*b*) the *arterial pile*,† in which the arteries, and (*c*)

* Given at the Medical Graduates' College and Polyclinic, November 18th and 25th, 1903.

† The chief artery in a pile may be as large as the radial.

Digitized by Google

the small *capillary pile*,* in which the capillaries predominate ; (*d*) 2 and 3 combined.

Most commonly a varicose condition of veins is

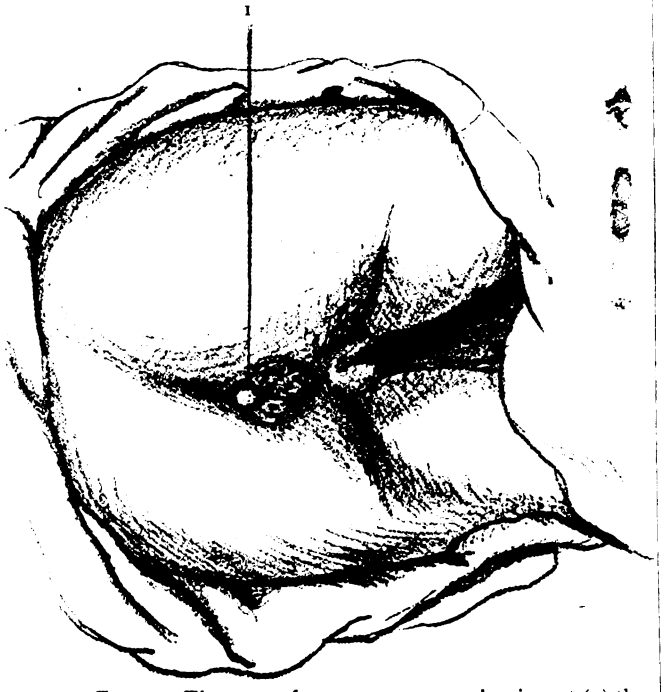


FIG. 1.—The anus of a young woman showing at (1) the sentinel pile covering the lower end of a fissure, and three ordinary external piles.

the chief factor in the production of piles, and varicose veins in the rectum are subject to the same accidents—thrombosis, ulceration, hæmorrhage, abscess, etc.—as varicose veins elsewhere.

Before we can definitely appreciate the abnormalities that we are called upon to deal with in the lower part of the rectum, we must be able to recall the salient features of the anatomy of the parts in which piles occur. These are the dilated lower part of the rectum, the *ampulla*, and the part within the clasp of the sphincters, the *anal canal*. The latter terminates above at the linea dentata, a zig-zag zone, the upward projections of which are termed anal papillæ, from which small ridges of mucous membrane, *columns of Morgagni*, are prolonged upwards, the relatively depressed intervals being the *valves of Morgagni* or *saccules of Horner*.

To show how necessary it is to recall these details if we are to understand what we see and feel in examining the lower part of the rectum, we need only remember that one of the commoner affections of this part, anal fissure, may begin by the separation of a valve of Morgagni which is torn down, like a "hang-nail" on a finger, till, swollen and deformed, it projects at the anus as a pale nodule, and is called a *sentinel pile* (see Fig. 1).

Another mental pause may be made before we pass to the clinical examination of the patient, and may be well occupied by answering the question, Where are the chief veins in the lower part of the rectum? If a healthy rectum is opened from behind and roughly dissected as it is removed in the mortuary, the chief veins in the ampulla and anal canal are seen to lie in three groups,* one anterior and two postero-lateral, as shown in Fig. 2. The arteries lies close to the veins. When the enlargement of the veins is great and wide-spread this arrangement is apt to be obscured, but when

FIG. 2.—The rectum of an adult opened in the mid-posterior line. The mucous membrane has been removed from the lower part to show the chief groups of veins.

* Capillary piles are "small, florid, raspberry-like tumours, or rather vascular areas upon the mucous membrane."—Allingham.

* The superior hæmorrhoidal veins (portal system) communicate freely in the anal region with the inferior hæmorrhoidal, belonging to the internal iliac group. Opposite the upper end of the anal canal tufts of small veins, probably remains of the vessels of the proctodeal membrane of the fœtus, enter the superior hæmorrhoidal veins.

Digitized by Google

the varices are not large the arrangement is clearly seen, as in Fig. 3. Internal and intero-external piles are illustrated in Fig. 4.*

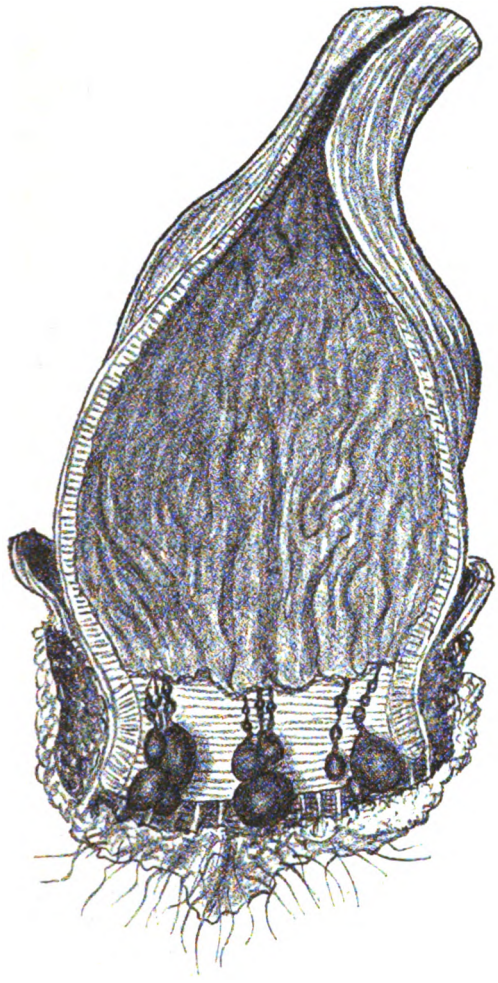


FIG. 3.—Thrombosed external piles. The thrombosed varices correspond to the three chief groups of veins. The white fibres of the longitudinal muscular coat are seen emerging below the internal sphincter.

These preliminary reflections having been made, we may pass to consider the examination of the patient. For an ordinary consulting-room examination, males may be examined standing with the body bent forward so that the head is a little lower than the pelvis. Females are best examined lying on the right side in the latero-prone position, with the hips and knees bent, the right arm behind the body, and the head on the same level as the pelvis. An operating table or a high couch is the most convenient; the surgeon can then sit with the part to be examined on a level with his eyes. If a speculum has to be passed the pelvis should be raised on a pillow, so that the rectum falls away from the speculum.

When, however, there is any difficulty in obtaining a complete examination, as is often the case in young women, or where there is any reason to suspect some more serious condition in the rectum, the examination should be made under anæsthesia, a good four-branched speculum, such as Allingham's, and a Kelly's rectal endoscope being at hand.

By digital examination internal piles are easily missed if the finger is passed up too far; only the tip of the finger should be beyond the anal canal, and then the lower larger ends of the piles are readily felt as the finger is rotated in the rectum.

Ordinary external piles appear as soft berry-like bodies; where thrombosis has occurred a firm, tender, bullet-like body is seen; such thromboses which have existed previously, and have undergone

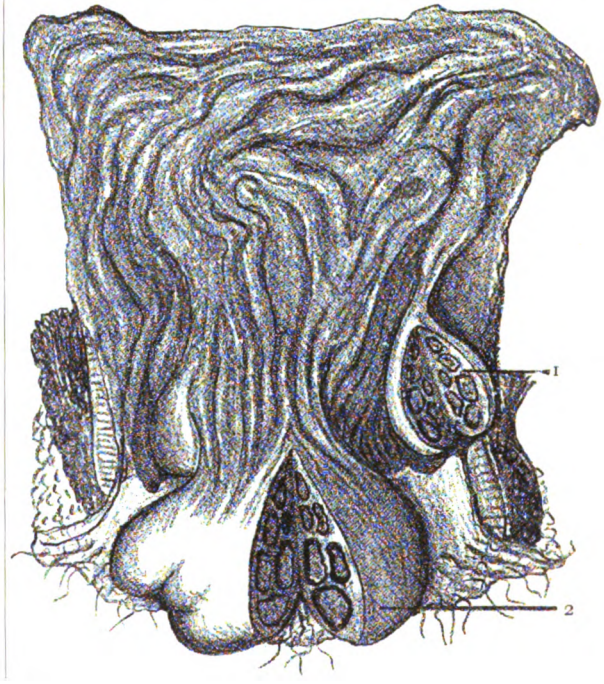


FIG. 4.—The lower part of a rectum, presenting (1) an internal pile which has been incised to show the dilated venous spaces occupied by clot; and (2) an intero-external pile. It will be noted that the attachment of "2" reaches the muco-cutaneous junction.

natural repair, leave the small tags of skin that are termed cutaneous piles. External piles cannot,

* Figs. 1, 3, and 4 are taken from the author's 'Surgical Pathology and Principles,' by kind permission of Messrs. Longmans, Green and Co.

Digitized by Google

from their anatomical position, be returned within the anus; whilst other things, such as internal piles, polypi of various kinds, and prolapsed mucous membrane, may be returned, and after being put back remain in that position when the patient is directed to contract and draw up the anus. Intero-external piles under the same circumstances can only be returned within the anus in part.

TREATMENT.—External piles are often only temporary engorgement of veins, and regulation of diet and tobacco, and regulation with a little local sedative and astringent* application, is all that is necessary. Patients often complain of uncleanness caused by external piles, which prevent complete action of the sphincter and allow some liquid to escape, and staining of linen follows. Or the complaint may be that it is difficult to cleanse the part after defæcation. In such cases, when the condition has been ascertained to be uncomplicated by internal piles, I think the most effectual way of dealing with the little varices is to dissect them out through a linear incision after dilating the sphincters. The upper and lower ends of the vein are twisted off and the incision carefully closed with fine catgut, all the precautions described below in connection with operations for internal hæmorrhoids being carefully observed.

For thrombosed external piles the treatment usually recommended is to slit up the little tumour with a narrow knife, turn out the clot, and pack in a little cotton wool to prevent the cut in the vein reuniting and the thrombus returning. This method is based on a supposition, which is not confirmed by my own observations, that the swelling is due to a localised extravasation of blood; it certainly has the drawback that occasionally much pain is experienced from spasm of the sphincter after operation; hence I prefer to dissect out the vein or veins, with their contained thrombi, after dilating the sphincters. This latter proceeding has the advantage of revealing any internal piles that may be present, and that should be removed at the same time. When thrombosed external piles occur, as they often do in women far advanced in pregnancy, palliative treatment, gentle laxatives, and locally a sedative application is the proper course to follow. We have to bear in mind that thrombosis is often a natural mode of cure, and that the obstruction to venous return in the superior and middle hæmorrhoidal veins, which exists during the later period of pregnancy, sets up a state of venous hyperæmia which militates against rapid repair.

Troublesome tags of skin may be snipped off in lines radiating from the anus, care being taken not to cut to the base of the tags, or too much contraction may result.

PALLIATIVE TREATMENT.—In the earlier stages of internal piles there is not much pain; the patient at most complains of aching over the sacrum. Hæmorrhage usually causes him to seek relief. Constipation will nearly always be present, even though the patient has a daily action of the bowels. Success will usually attend a course of Mist. Mag. Sulph. c. Ferro., and local treatment by the daily injection of ʒj of Liq. Hamamelidis. By these measures and a few days' rest in the horizontal position the piles may be cured. Before operation is decided upon, enlargement of the prostate and stricture of the urethra, and in the female displacements of the uterus or pelvic tumours, and in both sexes cirrhosis of the liver, must be carefully excluded. As in all operations, the urine should be examined, and the possibility of the patient being a bleeder should be eliminated.

The surgical treatment of internal piles, as at present practised in this country, resolves itself into removal by Salmon's method—combined incision and ligature,—though some surgeons still prefer the clamp and cautery. Accidents after either method are rare, but though rare they should never be left out of consideration. It is claimed by those who exclusively use the clamp and cautery that there is less pain than after the operation by ligature; but in my experience, if the sphincters (note the plural) have been completely and sufficiently stretched, the pain after operation on piles is very slight.

PREPARATION OF THE PATIENT.—Many troubles are avoided by careful preparation of the patient for rectal operations. It is of advantage if three days can be given up for this purpose. During this time, for the slighter operations, such as piles, the patient may have a full but light diet, *e. g.*:

* I avoid the prolonged use of lead lotions owing to the possible absorption of a harmful quantity of lead into the system. The same applies to strong preparations of belladonna. The ointment, Calomel, gr. ij; Bismuth. Subnitrat., ʒij; Cocaine Hydrochlor., gr. iv; Vaseline, ʒj; is safer.

Digitized by Google

Breakfast.—Toast, butter, egg, boiled fish, weak tea.

Lunch.—Chicken or fish, mashed vegetables, junket with baked apples, toast.

Tea.—Weak tea, brown bread and butter.

Dinner.—Boiled or roast mutton, lamb, chicken or other light meat, with mashed vegetables; custard; toast and butter. Rice, hot spiced foods, and alcohol should be avoided.

For three successive nights Pil. Hydrargyri gr. ij, with Pil. Colocynth et Hyoscyami gr. viij, in two pills in the case of an adult, followed, if the action of the pills is not severe, two hours before breakfast next morning by a full dose of saline, *e. g.* ℥ij Mist. Mag. Sulph.:

Magnesii Sulphatis . . .	℥j.
Magnesii Carbonatis . .	gr. xv.
Aquæ Menthæ Piperitæ . .	℥j.

On the morning of the operation a tumbler of warm milk, followed by ℥j of Mist. Catechu Opiata:

Tincturæ Opii	♏x.
Tincturæ Catechu . . .	♏xv.
Liquoris Ammoniæ . . .	♏v.
Tincturæ Cardomomi Co. . .	℥ss.
Aquæ Menthæ Piperitæ .	ad ℥j.

should be taken four hours before the operation. Chalk mixture is to be avoided, since it is liable to cause scybala. Two hours before operation a good enema should be given, and after it has returned the anal region should be cleansed and shaved, and an antiseptic dressing of 1 in 40 carbolic lotion* applied.

Stretching the Sphincters.—For this important preliminary to many operations on the rectum the patient must be fully anæsthetised, ether being preferable, unless some definite contra-indication is present. Both thumbs are inserted into the rectum, and they must be felt to pass above the internal sphincter. Gently and gradually the thumbs are separated, first in an antero-posterior direction, till the pubes and coccyx can be felt at the same time. This stretching is then repeated in a lateral direction, till the inner aspects of the tuberosities of the ischia are felt. These processes are repeated till all tendency to contract is abolished, and the stretching should be so gradual that no laceration of the mucous membrane is produced. In elderly patients, after the gradually yielding muscle has been overcome, a firm fibrous ring may be felt; it is undesirable to overcome its resistance, even though the stretching has to stop short of the bony boundaries mentioned above. My rule is to allow ten minutes to the stretching; in this way the muscle will be rendered passive for two or three days, after which its power of contraction gradually returns, but without spasmodic action. Nearly all the pain that follows operations for piles is due to insufficient stretching of the sphincters.

The Operation by the Ligature.—The patient may be either on the right side or in the lithotomy position. After complete stretching of the sphincters, the lower three inches of the rectal mucous membrane are exposed to view, and the position of the piles can be studied. The commonest position for an internal pile is, especially in women, anterior—the perineal pile, as it is termed. Next to this the postero-lateral are most common, but cross branches of veins may be enlarged, causing piles in the intervening spaces. It is important to study the disposition of the piles and so plan the incisions that the subsequent contraction of the mucous membrane will not be excessive. The smaller piles should be tied first, or they may become obscured by blood and overlooked.

The pile to be removed is seized at its most prominent part by a pile hook, and drawn down and away from the bowel wall by the surgeon. The whole of the lower attachment of the pile is then separated by a cut made with scissors parallel to the surface of the coats of the bowel. The circular muscle of the bowel should not be exposed, but the incision should leave the deeper part of the submucous tissue. If the pile is purely internal, the lower limit of the cut will be at the upper part of the mucous lining of the anal canal; if intero-external, it will be between the skin and the lower limit of the anal canal. The ligature should be of sterilised plaited silk, so stout that it cannot be broken by a steady pull, and not too thick to tie easily. Whilst the ligature is being placed as high as possible at the upper attachment of the pile, the latter is drawn steadily down by an assistant, but not so forcibly that there is risk of including the muscular coat of the rectum in the ligature.

* Strong (1 in 500) sublimate lotion is to be avoided in this region.

Digitized by Google

The ligature is tied as tightly as possible, and after a good reef-knot has been tied a third hitch may be made for extra security. It is exceptional for any vessel other than those entering the pile from above to require ligature, but should an artery be seen spouting, it should be caught up with catch forceps and tied. The bulk of the pile below the ligature may be cut off, if enough of it can be left to prevent slipping of the ligature.* After all the piles have been tied, and the condition of the patient is good from the anæsthetist's point of view, a few points of fine sterilised catgut suture may be inserted to draw together the edges of the mucous membrane below the tied stump of the piles. This will often accelerate repair.

The Dressing.—I wrap a strip of thymolated iodoform gauze lightly round my index finger, the tip of which is allowed to protrude, and, after coating the exterior with iodoform ointment, gently pass it into the rectum so that it rests against the wound surfaces. The finger which is coated with ointment is gently withdrawn, and a small but firm piece of drainage-tube inserted in its place, two or three inches protruding below. This is packed round with absorbent wool, which is applied firmly with a T-bandage; in this a hole is cut to allow the end of the dainage-tube to show, and a second covering of wool is lightly applied. The tube allows the ready escape of flatus, and gives early warning of hæmorrhage should this unusual and undesirable event occur.

After-treatment.—Retention of urine is commoner in male than in female patients. It may be due to pressure of the dressing on the perinæum, so the bandage should be loosened before resort is made to the catheter. Hot fomentations above the pubes often assist the patient, who may be allowed to kneel in bed and attempt urination in that position. If these measures fail, a No. 8 rubber catheter should be boiled in water (not soda solution); the meatus of the patient and the hands of the surgeon are cleansed with 1 in 40 carbolic, and the catheter passed preferably without a lubricant, but if a lubricant is necessary, recently boiled olive oil should be used. In order to avoid cystitis all this care must be taken in passing the catheter as long as the retention lasts, and this may be for fourteen days or more; but it usually ends with the first action of the bowels, if not before.

Confining the bowels. — If the patient has been prepared as described above, no morphia, either in a suppository or in hypodermic injection, will be required immediately after the operation, which should be done early in the day. A morphia injection may be given in the evening, or earlier if there should be any pain, and on the succeeding three days the Mist. Catechu Opiata will be required three times daily. The diet should be light—soup and milk at first, then milk, gruel, and toast; eggs and minced chicken may be added after the third day. Save in emergencies, alcohol should be avoided.

Dressing the wound.—Twenty-four hours after the operation the tube should be removed, and the gauze irrigated with warm boracic lotion and removed. After washing well with the lotion a little iodoform ointment, followed by a boracic fomentation, is applied. Gentle irrigations with warm boracic lotion containing Acidi Borici ʒijss, Solutionis Thymol Oj, should be used whenever the fomentation is changed.

Opening the bowels.—Two of the mercury, colocynth, and hyoscyamus pills should be given (for an adult) on the third or fourth night, followed by a good saline—*e. g.* ʒij sodium sulphate in half a tumbler of warm water—the next morning, and if there is any tenderness ʒiij of warm olive oil should be injected as soon as the bowels promise to act. Daily actions should be secured by aperients, of which cascara sagrada (ʒss liq. extract) is one of the best.

Convalescence.—A minimum of a week's recumbency is required. The ligature usually separates about the eighth day, and before the patient is allowed out of bed the condition of the rectum should be ascertained. If any ligatures have not come away, they should be removed by gentle traction. If too much contraction of the anal canal is taking place, it will require the daily passage of the surgeon's forefinger well anointed with iodoform ointment. The patient is usually cured in three weeks from the day of operation. After this he should be taught to strengthen the sphincters and levatores ani by daily exercises—ten to fifteen rhythmical voluntary contractions being made night and morning.

* The part left should be sufficiently long to prevent retraction of the artery, which may otherwise escape from the ligature and cause hæmorrhage.

Digitized by Google

TWO LECTURES
ON
BRONCHIECTASIS.

Delivered at the Hospital for Consumption and Diseases of the Chest, Brompton, October 21st and 28th, 1903.

By S. H. HABERSHON, M.D., F.R.C.P.,
Physician to the Hospital.

LECTURE II.

GENTLEMEN,—In my last lecture I endeavoured to give you as complete a clinical picture of bronchiectasis as was in my power, and I think you will say that a very fine group of cases was presented. I was exceptionally fortunate in having at my disposal so much material in the hospital at the present time. These twenty cases, I think, showed most of the chief characteristics, but it is quite obvious to you that one part of the clinical picture of the disease I could not show in a lecture room, and that part has reference to the more acute stages and to many of the more serious complications that take place. To begin with, bronchiectasis occurs at any age; it may occur in very small children—that is to say, in infants,—or it may occur in people of extreme old age. The commonest age, however, is in early and middle life. The largest number of cases are in the age period from twenty to forty. If you look at Laennec's earliest description of the disease, you will find that he describes several cases in young children, one an acute case in a little infant, beginning with whooping-cough, death occurring within six months from the onset. He then describes the case of an old lady of seventy-five who had had the disease for fifty years. He also describes the case of a man aged forty-one, who had had the disease for twenty-five years, death having ensued at the end of that period. So you see the disease may run an extremely rapid and acute course, or it may be prolonged for a very considerable proportion of the person's life. When it occurs in little children you will remember that it is often mistaken for attacks of vomiting; the paroxysmal attacks of the disease are exceedingly like those of simple vomiting, the sputum all coming up in one gush. Except for those very acute cases which we know as bronchiolectasis, the disease as a rule runs a very chronic course. But one exception to that I must make, and that is in the case of foreign bodies in the bronchus. Those cases which are caused by a foreign body in the bronchus are very rapidly progressive, and I shall have occasion to read you the notes of one such case. But in spite of the chronic nature and the comparative well-being of many of the cases, they are liable to certain complications, and these I have put down on the syllabus which has been distributed among you. *Acute inflammatory attacks* are extremely common. *Acute broncho-pneumonia*, *acute bronchitis*, especially in the cylindrical cases, and *acute pneumonia*. Then sometimes, owing to the decomposition which goes on in the sputum, and the consequent ulceration of the bronchi, all kinds of *septic attacks* may occur. These septic attacks I have mentioned on the paper as including *gangrene of the lung*, *septicæmia*, *pyæmia*, and sometimes *abscesses* in different parts of the body, and most commonly in the brain. Sometimes also there may be abscesses in the liver. There may be empyemata, and occasionally complete ulceration through the pleura, forming a pneumothorax. Or there may be some of the other accompaniments of pyæmic attacks, such as pericarditis or pleurisy. Then a third class of cases are those in which a very large area of the lung is involved, and therefore you get a great stress thrown upon the heart. You have the incidents of *cardiac dilatation*, and eventually dilatation of the systemic veins. The features of the case then would be no different from the last stages of a case of long-continued pulmonary fibrosis or of emphysema. Then one cause of termination in these cases is that the patient becomes exhausted, either from the large invasion of the lung or else from the toxic effects, so that though many of the patients may have these septic symptoms, still the actual death will take place from exhaustion. Lastly, there is a complication of the disease which does not necessarily compel the patient to remain in bed; I refer to certain peculiar joint affections. Now this particular joint affection is known by the name of pulmonary osteo-arthropathy. You know of all the osteo-arthropathies, some are inflammatory and hypertrophic, and others depend on atrophic changes, and are nervous in origin—such, for instance, as the osteo-arthropathies of locomotor ataxia and other spinal diseases. The present cases differ from these in that the greater part of them are hypertrophic in character,—that is to

Digitized by Google

say, there is a new growth of bone, or of periosteal thickening. The disease was first described by Marie in his well-known article on acromegaly, and he pointed out that in certain cases of lung disease, especially chronic lung disease, the joints tended to become affected. Sometimes the invasion of the joints is quite painless; at other times acute attacks occur, with which there is a good deal of aching; but the pain is not such a prominent feature as in the arthritis of rheumatoid arthritis. This disease not only occurs in bronchiectasis; a chronic case of empyema, and many other conditions of the lung, such as chronic pulmonary tuberculosis, may show this complication. The joints most commonly affected are the wrists and ankles. Sometimes the knees also are involved, and when you come to investigate by our more modern methods, by the X rays, it is found that there is a deposit of new bone upon the surfaces of some of the long bones also. The metacarpals and sometimes the first phalanges and the radius and ulna, as well as the tibia and fibula, are affected. I shall be able to show you certainly one, and possibly two cases of this disease. I have also a considerable number of radiographs, and I think it will be better for me to show these together with my specimens and cases. A few cases show no sign of bony enlargement in the radiographs.

With regard to the CAUSES OF DEATH, *exhaustion* perhaps covers a large number of cases. There is also *cardiac failure*, and sytemic engorgement of venous circulation; *abscess of brain*, and other *septic and inflammatory conditions;* and lastly, *hæmoptysis*. Now there are many published statistics, but I thought it would be wise to try and see whether our own post-mortem records corroborate those already known. I have been through about 2000 cases of autopsy, covering twelve years of the Brompton Hospital records. Out of these 2000 cases there were 64 cases of death from bronchiectasis,—that is to say, in 3·2 per cent. of all autopsies on chest cases the deaths were from bronchiectasis. In 60 cases where the sex was stated, 43 were males and 17 females, so that rather more than two thirds of the number were males. The average ages of the patients were as follows (I have not been able to find out the ages of all of them, because in some it was not recorded; I found the age mentioned in some 26 cases, and it bears out what I said just now, that it is particularly a disease of middle life):—Under twenty years of age there were 6 cases; between twenty and forty, 12 cases; and over forty, 8 cases. I think that is a very fair example of the ages at which the cases occur. Of the causes of death, *exhaustion and cardiac failure* were responsible for 20, but these included all the cases in which bronchiectasis was associated with pulmonary tuberculosis. I think there were something like 15 or 16 cases of pulmonary tuberculosis included in these 20. Two died after an operation for a dilated saccule, and 1 of lardaceous disease. Now *septic cases* form a very large proportion of the whole list. Of *septic pneumonia* and *gangrene* of the lung there are 11 cases; *septic pericarditis*, 2 cases; *pyæmia and abscesses of the liver*, 3 cases; *empyema*, 3 cases; *abscess of brain*, 7 cases; and 1 case of *pneumothorax*. The third cause was *hæmoptysis*, which was the cause of death in 4 cases. There were 10 cases in which the causes of death were some *inflammatory* condition—namely, 6 cases of broncho-pneumonia and 4 cases of acute bronchitis. In order that I may try and impress the acute complications that occur, I should like, in the briefest manner, to relate 3 fatal cases that have recently been in this hospital.

The first was that of a patient, J. F—, æt. 45, a porter. He had influenza twelve months ago,—that is to say, in June, 1902. He had had cough and foul-smelling expectoration since January, 1903, and clubbing of fingers was observed. There was pain in the left side of the chest, and some emaciation and loss of strength. On admission on June 24th, the left chest was found to be dull, with blowing and weak amphoric breathing, with pectoriloquy, and bubbling râles over the left upper lobe. The temperature on admission was a little irregular, but rarely over 99°. Sputum about six ounces per day, and was offensive. On July 7th and 8th the patient complained of pain in the left side. A friction rub was heard on the 9th. The temperature rose on this day to 102·6°, and at the same time the sputum increased to an average of about ten ounces. The temperature from this time onward become more irregular, rarely reaching the normal, the sputum becoming more offensive. In August the patient became much worse, with a high irregular temperature—100° in the morning and 102° or higher at night, with profuse sweats. There was considerable drowsiness, and on August 15th it was noted that he was in a somnolent condition.

Digitized by Google

Optic neuritis was found, but no localising signs of any cerebral abscess. The patient died on August 18th. At the autopsy thirty-three cerebral abscesses were found in the cerebral hemispheres.

The second case is that of a footman, H. M—, æt. 20. He had pleurisy and pneumonia in the right side. At the age of sixteen he was in St. Thomas's Hospital. He had two other attacks of pleurisy—namely, at the ages of fourteen and seventeen. He was never tapped. At seventeen years of age he was at the Ventnor Hospital for fourteen weeks on account of hæmoptysis, and had profuse expectoration of blood and matter mixed, about three ounces in one week. He recovered from this and felt quite well. In August and September, 1902, he suffered from a cough, then had another attack of pleurisy on both sides after getting wet, and was ill until his admission on August 17th this year. The cough became more paroxysmal on the previous May, and two or three times a day he would bring up about an ounce at a time of offensive mucus. On admission the expectoration was less in amount but very offensive, more in the morning than at other times. The breath was offensive, and fingers clubbed. The physical signs were exceedingly slight. No impairment of resonance; weak breath at both bases. Behind there were some grating râles. The disease pursued a normal course, and he improved until September 18th, when the breath became short and the temperature began to rise. On September 22nd an attack of great pain was experienced on the right side, near the nipple, with marked friction. Temperature 102° to 104°. There was impairment of respiration at the base, with bronchial breathing, and the attack was considered to be pneumonic. Death ensued on September 30th. The post-mortem showed general cylindrical bronchitis in both lower lobes, and right upper and middle lobe; there was also a diffuse pneumonic change in the right lower lobe. I have selected these cases because they are very typical of the sudden onset of pneumonic symptoms.

The last case I wish to mention was that of J. W. S—, æt. 51. In March, 1902, he was in a great hurry to eat some Irish stew, and suddenly a piece of bone went the wrong way. He went to a doctor, who, the patient says, passed an œsophageal bougie, because it was not clear whether the bone had gone down the œsophagus or into the trachea. However, nothing could be found, and he was examined with a laryngoscope, but nothing was discovered. He went on with his work until August of this year. He was complaining of a cough which he had had since March, 1902, and offensive sputum. He brought up a pint of extremely offensive sputum each day. Several consultations were held about his case, and he saw a surgeon, when the question was raised as to whether an operation should be carried out. Both Dr. Hugh Walsham and Dr. Mackenzie Davidson took radiographs of his chest. On one occasion nothing was found, but on another two doubtful spots were seen in the right lung. He was taken into this hospital, and was very ill. On the second day after admission there was severe pain in the right side, and on the following day signs of pneumothorax were detected. He was aspirated, and about seventeen ounces of pus with some air were drawn off. He died from collapse a few hours later.*

I must now leave the cases and turn to the question of PROGNOSIS. In the early stages, and without any coincident lung complications, the prognosis may be exceedingly good. Sometimes in little children, where there is an acute invasion, the bronchial tubes will recover themselves, and I think you will more easily understand that when we come to deal with the pathology of these acute cases. Then in the more chronic cases, provided there is not a very large accompaniment of chronic pneumonia, the case may go on for an exceedingly long time. At the same time, where there are many saccules, and above all where the sputum is excessive and very offensive, there are great dangers, and the prognosis is exceedingly difficult to give. Even the cases I have just read to you illustrate how suddenly complications may occur. A case may be going on apparently pretty well, when suddenly an attack of pneumonia or a septic attack may appear. *Excessive fœtor*, when the sputum has been almost free from fœtor, introduces an element of danger, showing that there is great liability to septic complications. All *acute febrile conditions* should arouse our anxiety in a chronic case. *Hæmoptysis*, if it is at all excessive, is dangerous, and in one or two of the specimens I

* I am indebted for permission to publish these brief notes of cases to my colleague, Dr. Kidd.

Digitized by Google

show you here you will see that aneurysms do occur, and also ulcerated vessels. If you look at the syllabus I have given you, you will see I have summed up the cases, and I have given four different incidents that influence prognosis: *the extent of lung tissue involved* and the number of dilated bronchi; the *fœtor of the expectoration*; the *inflammatory and septic incidents* of the disease, which are always liable to occur in advanced cases; and *hæmoptysis*, if excessive. Pneumothorax, empyema, and any cerebral symptoms are dangerous incidents.

I next come to the PATHOLOGY of the disease. There are generally described two forms of the disease—the *cylindrical* or *fusiform*, and the *sacculated* or *globular*. I shall be able presently to illustrate the differences between these two from the large number of specimens that you see upon the table. Besides these two forms you will often find a combination of the two present. Now the general pathology is associated with two conditions of bronchial tubes. Sometimes the tubes are atrophied, but more commonly they are hypertrophied. Atrophy of the bronchial tubes is more often seen in those cases where there is emphysema; but where the disease is due to previous inflammatory attacks, hypertrophy of the bronchial tubes is the commoner occurrence. Now will you picture to yourself the distribution of connective tissue and fibroid elements in the lungs? Suppose we were to eliminate all the epithelial structures, we should then have a supporting framework which would consist of the muscular, fibroid, and elastic walls of the bronchi, continuous with the interlobular and interalveolar septa and connective tissue. On the other hand, supporting the external walls you would have the subpleural tissue which sends its delicate strands and fibrils connecting it with the interlobular and interalveolar network. So that if you were to take away all the intervening epithelial tissues, you would still have a spongy, as it were, supporting structure. Now in many of the cases which depend upon inflammatory attacks the chronic inflammation leads to a fibrosis, spreading either from the bronchial tubes or from the pleura. For instance, you have a peribronchitis. Incessant attacks of bronchitis, as in all other irritations in the body, cause proliferation of tissue. You have not only an invasion of small cell growth into the bronchial wall, but you eventually have a great increase of the fibroid tissue of the bronchi. That may spread, especially if it is accompanied by a coincident broncho-pneumonia, to the tissues between the alveoli and between the lobules, leading to an interstitial fibrosis. The same process may commence from repeated attacks of pleurisy, and you will see in some of these specimens distinct bands passing from the pleura. In similar manner this chronic inflammatory process, proceeding from without, extends to the interstitial supporting framework of connective tissue and fibrous elements of the lungs, which eventually may be enormously increased, so that a dense cirrhosis of lung is produced. In addition to this interstitial change there is a second process by which the lung becomes consolidated. The disease is accompanied in pneumonic cases by attacks of broncho-pneumonia or lobar pneumonia, and these lead to an organisation of the contents of the alveolus. By organisation I mean that not only is the alveolus filled with new cell growth, but you have definite vascularity with development of new loops of blood-vessels, so that a section, especially if double-stained with eosine (which picks out the blood-vessels), shows the alveolus full of cell growth —often of fibroid cells,—in the midst of which numerous sections of capillaries are interspersed. I have under the microscope a section which will illustrate this.

The gradual inflammation and dilatation of the bronchi very speedily destroys the ciliated epithelium, but you will generally find the basement membrane left behind. In the normal bronchus, underneath the basement membrane, there is the alveolar tissue and a layer of elastic fibres; and beyond that a narrow layer of unstriated muscular fibres, and then your cartilage plaques in the larger bronchi, surrounded by a certain amount of fibroid tissue. The muscular and elastic tissue, when there is atrophy of the walls of the bronchus, are thinned out, and practically can only occasionally be seen. In the hypertrophied variety the muscular tissue after a time not only becomes destroyed, but is converted into fibroid tissue, so that you cannot find any trace of the muscular fibres. I have two specimens here of a wall of the bronchus, one of which is especially stained to show the elastic tissue and another the fibrous tissue changes. Of course, in cases where there is ulceration of the bronchus the whole wall may be destroyed. The general macroscopic appearance of a dilated bronchus is

Digitized by Google

that its wall is more thin and velvety than usual, but sometimes smooth and extremely vascular. I think these are the main points. I would just say that the fibrosis of lung varies very much in different cases. Sometimes interstitial fibrosis preponderates, and in those cases very often you will see bands stretching across the lung, and even the interlobular bands extremely thickened, giving a strikingly tesselated appearance. The interstitial change and the organising pneumonia are most commonly coincident. Before I come to show the cases which are here, and the photographs and specimens, I should say one or two words about treatment.

The treatment of a case of bronchiectasis in the first place is *general and hygienic*. Tonic treatment should be carried out by means of drugs and food and stimulants, iron, quinine, and cod-liver oil and glycero-phosphates. The patient should be warmly clad and protected from any incidents which will make him liable to bronchitis or pulmonary attacks. Climate has a great deal to do with that. If the patient is well-to-do or rich, he can be sent away to a drier climate. Of course some of the best places are Egypt or California and certain parts of South Africa, the Karoo district, and many other places. In the case of our hospital patients we are not able to alter the climatic conditions for them, but where possible a dry, equable climate, which is not too humid, should be advised. The *local treatment* is mainly to *induce free expectoration* on the one hand, and to *lessen the fœtor* of the expectoration. To make expectoration more free we give alkalies internally and such drugs as ipecacuanha, small doses of iodide of potassium, creasote, and guaiacol. These are most valuable. In this hospital the creasote treatment has practically superseded many of the other modes of treatment. Then we give inhalations that are *disinfecting and deodorising*, such as carbolic acid, menthol, thymol, oil of peppermint, pine-wood oil, terebin, and eucalyptus. Internally we give astringents where there is much chronic catarrh, and drugs to promote free secretion. I have mentioned ipecacuanha and alkalies, but benzoic acid and the various balsams and guaiacol are of great value. One form of treatment has been tried a good deal in this hospital, the treatment of giving garlic to diminish the fœtor. Garlic is often given, either chopped up or as an oil in ½ to 3 m, three times daily in capsules, and this latter is perhaps one of the most pleasant methods of giving it. Some people rely very much on *intratracheal injections*, and one of the most valuable is a drachm of two parts of menthol and two parts of guaiacol and eighty-eight of olive oil. There is another method of treatment used in this hospital, that of hypodermic injection. Dr. Maguire injects a solution of formic aldehyde directly into the veins. He believes, and I think truly, that it diminishes the fœtor of the expectoration. The treatment of acute and chronic catarrh is based on general measures. And we need not enter into detail about the treatment of other complications. For hæmoptysis, if it is small, we give astringents. If it is very large, we take measures to reduce the blood-pressure, such as giving the patient a small amount of fluid and the internal administration of calcium chloride. If the hæmoptysis is large, this chloride of calcium should be given heroically; I mean half a drachm every two or three hours until the hæmoptysis ceases. With regard to the surgical treatment. It has been tried on several occasions in this hospital, but the results have not been good. You will remember how many saccules you have to deal with, and there was a case here which was opened under the idea that there was only one saccule, because the physical signs seemed to point to that. Where you have septic conditions set up I think it is justifiable to resort to operation, but it is difficult in bronchi, which exist at so many varying depths, to be quite certain that you have to deal with only one saccule.

I will now show you a series of microscopic specimens which illustrate the varying conditions in the bronchial walls; also a large group of specimens from our museum.

Further, I have to show you a case of chronic pulmonary arthropathy, and I shall add to this case a series of radiographs showing the changes in the joints and long bones in several other cases.

Soxhlet's Sugar Food in Infant Therapeutics.—Brüning states that his experience with this substance, added to the milk, has been extremely favourable in the case of thirty-seven infants, some of them very sick. Even those with severe gastro-intestinal trouble gained in weight as well as the others on this diet.—*Journ. A. M. A.*, December 5th, 1903.

Digitized by Google

A CLINICAL DEMONSTRATION

At the Medical Graduates' College and Polyclinic,

By HAROLD L. BARNARD, M.S., F.R.C.S.

GENTLEMEN,—These first two cases which I show you this afternoon are examples of different varieties of *rannulæ*. The first is in a woman who suffered from syphilitic scarring of the floor of her mouth, and this led to stenosis of the openings of both Wharton's ducts, so that the ducts behind the obstruction became dilated into cysts. At one time it was supposed that all rannulæ were produced in this way, but subsequently it was found that a probe could be passed along Wharton's duct in most cases, and that this probe did not enter the cavity of the rannula. The present case is therefore an example of the older pathology, and the other, which I am about to show you, arose from a cystic degeneration of one of the buccal glands in the floor of the mouth, which is now held to be the pathology of most rannulæ.

This little boy, J. F—, æt. 12, came to the London Hospital with a typical rannula unconnected with Wharton's duct,—that is to say, a probe could be passed along this duct without entering the rannula. Beneath his tongue there bulged a bluish little cyst just to the left of the middle line, over which coursed several dilated veins. An effort was made by a colleague of mine to dissect out the entire cyst, but, as so often happens in these cases, parts of the delicate wall of the sac were left behind, and became encysted in the floor of the mouth. Three months later, when the boy came under my care, the whole condition had apparently relapsed. I made a second effort to cure him by removing the mucous membrane of the floor of the mouth over the cyst with the adjacent half of the cyst wall, and then hemming with catgut the edge of the cyst and mucous membrane together, so as to freely lay the cyst open into the buccal cavity. This operation was apparently successful as far as the floor of the mouth was concerned; but he subsequently developed this large, diffuse, and soft swelling beneath his chin, which is apparently an extension of the myxomatous material of the rannula through the floor of the mouth into the subcutaneous tissue of the submental region. As the swelling causes the boy no inconvenience, and is a source of income to him, he naturally refuses any further surgical interference. This case well illustrates the great difficulty which is often experienced in eradicating these little cysts from beneath the tongue. But I have never before seen one which was driven through the floor of the mouth.

The next case is one of *gummatous bursitis of the subpatellar bursa* of the right knee in a woman of forty years of age who has had several miscarriages. The swelling, you will see, is deep purple in colour, and extends not only beneath and on either side of the patellar ligament, but also spreads in the subcutaneous tissue on either side of the knee-cap, where it is ulcerated in a serpiginous manner typical of tertiary syphilis. The case is improving rapidly on potassium iodide and perchloride of mercury. This case serves to illustrate the traumatic origin of gummata. You are all aware that the most frequent site of gummata in men is on the shin, where they are most exposed to injury; whereas in women belonging to this class gummatous affections are most frequently seen around the knee-joint, because in carrying out their household work they have to do much scrubbing and kneeling. The best illustration of a traumatic gumma that I have encountered was in a woman who received a blow on her forehead. This was followed by a typical hæmatoma, and when she attended my out-patients' I told her it would subside in a week or so; but at the end of a fortnight, instead of diminishing in size, it was increasing, and had become purple, and appeared about to perforate the skin. On inquiry I found she had a syphilitic history, and a few doses of iodide of potassium served to dissipate the swelling.

This is a case of *gummatous synovitis of the knee-joint* in a woman of fifty-four, which I brought here to-day in order to contrast it with the last case, but I fear that the cure has been so effectual that it is not a very good case for demonstration. It will serve, however, to illustrate the point that tubercle tends to attack the two ends of life and syphilis the middle. Tubercle as a primary lesion commences almost always before thirty or after sixty, and therefore, if we encounter what seems to be a tubercular lesion commencing between the ages of thirty and sixty, we should always consider whether it may not after all be syphilitic. In the present case of this woman the right knee-joint was filled with fluid,

Digitized by Google

the synovial membrane was thick and pulpy as it is in tubercular synovitis; but the disease had commenced at the age of fifty-four, and with her past history there was little doubt that the condition was one of gummatous synovitis. She attended my out-patients' from November, 1902, to January, 1903, and on mercury and iodide the swelling of the knee-joint very largely subsided. But you will notice that the knee-joint has been more or less seriously damaged by the disease, so that now it is loose, and there is considerable creaking and grating on flexion. Indeed, it is worn out before its time—a condition which in surgical language would be called rheumatoid arthritis.

Our next case is one of *keloid arising on four vaccination marks*, which I owe to the kindness of Mr. James Sherren, who has lent it to me to show to you. Each vaccination mark is raised above the surrounding skin like a little button, and these swellings are smooth, elastic, firm, and pink. This condition depends upon a constitutional tendency of the patient, and has very little to do with the character of the injury which produced the scar. The pathology is important, because it clearly indicates that these cases should be left alone, and that they will ultimately get well of themselves. When an injury is inflicted on the skin blood is poured out, and this, as you are well aware, is removed by leucocytes from the neighbouring vessels. Then in every wound proliferation takes place in the neighbouring connective-tissue cells, especially in those of the capillaries and lymphatic vessels. The product of this growth is a vascular mass of tiny spindle-cells, which under the microscope very closely resembles spindle-celled sarcoma. However, in the course of a week or two, in ordinary scars, this swollen vascular tissue becomes pale and shrinks; and this change in the macroscopical appearances is due to the microscopical spindle-cells, which are known as fibroblasts, manufacturing in the spaces between them long strands of white connective tissue, which shrink, contract, and obliterate the capillaries. In people with a keloid predisposition the swollen pink fibroblastic stage of scar-formation is prolonged and exaggerated, but if we have patience and wait, the tardy fibroblast will ultimately form the desired fibrous tissue, and the scar will become white and contract. Some keloids appear to arise without any cause upon the chest, and when they do so they sometimes spread extensively and progressively. In such a case which I saw about two years ago I was able to definitely settle that the starting-point was a minute patch of lupus vulgaris, and this explained the progressive character of the keloid. For the lupoid edge irritated the tissues of this patient, who was predisposed to keloid, and produced immediately the swollen keloid mass, which in the course of a year or two went on to contract and form white fibrous tissue. The patient pointed to a tiny spot of lupus in another part of his chest, and told me that that was how the original patch of keloid commenced. I cannot, of course, say that this explains all cases of idiopathic progressive keloid. It is claimed by some people that thyroid extract will disperse these keloid masses, especially in tubercular subjects; but although I have seen this remedy applied on several occasions, I have never seen it do any good.

The next case is one of a little girl of fifteen, with a *parenchymatous enlargement of her thyroid gland*. On the same afternoon that she came to my out-patients' at the London Hospital a young Jewess of the age of twenty-one attended with a large adenoma in the right lobe of her thyroid gland; and these two cases together serve well to illustrate the signs, the treatment, and the ultimate result in these very different conditions of the thyroid gland. In this little girl you will see that the enlargement is symmetrical, both lobes are equally enlarged, and the isthmus is correspondingly increased in size. The larynx and trachea as a consequence are strictly in the middle line, and if you feel this thyroid gland you will find it smooth in outline and homogeneous in texture; whereas in the case of the young Jewess the swelling was restricted to the right lobe of her thyroid, which was as large as an orange. The isthmus was not involved, and the trachea and larynx were pushed over on to the left side. Moreover the tumour fluctuated, and was much more elastic than what could be felt of the left lobe of the gland. The treatment also was a contrast in these two cases. This little girl was placed upon one tabloid of thyroid extract daily, and this was increased up to three. In the course of three months the circumference of her neck has diminished from 12½ inches to 11¼ inches, and the appearance of her neck now approaches the

Digitized by Google

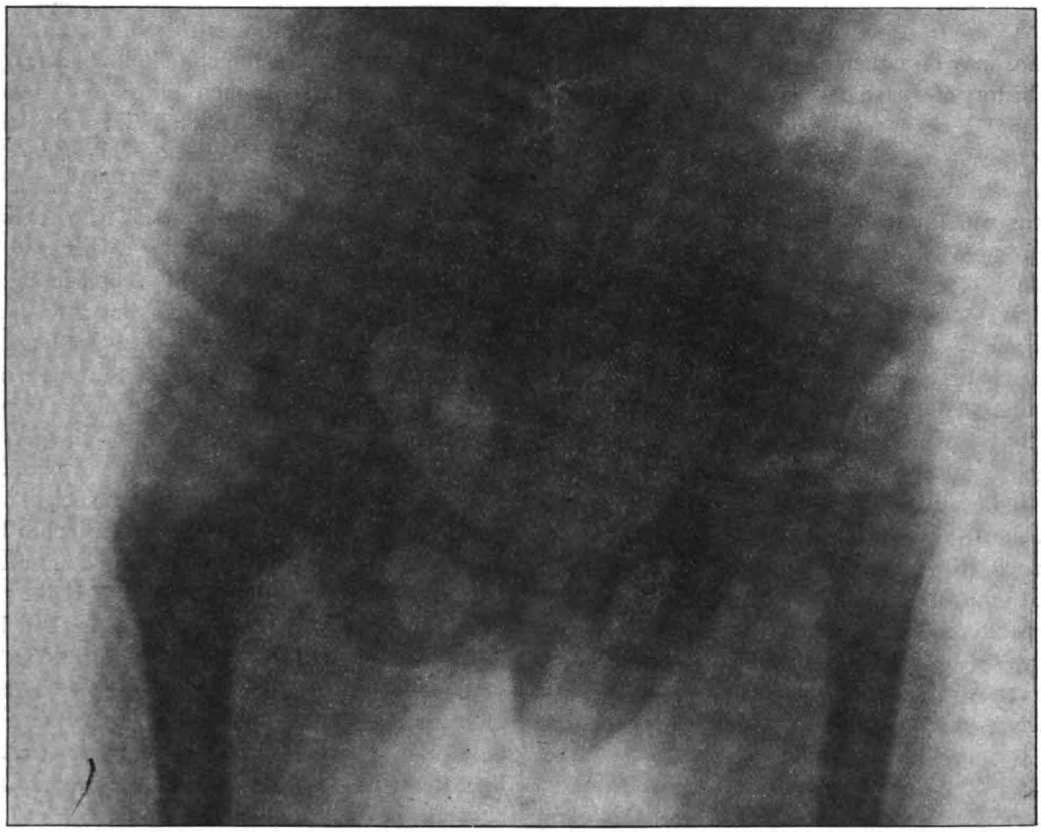
normal. I might with advantage have prescribed in addition Pot. Iod. gr. v, and Tr. Iod. ♏v, in a tumbler of water twice or thrice a day, as is recommended by Mr. James Berry. On the other hand, we informed the young Jewess that, as the adenomatous tumour was pressing slightly upon her trachea, it must be removed, and that this could only be done by an operation. The best operation for adenomatous cases is enucleation, for this operation is attended by an extremely low mortality. We accordingly enucleated the tumour from the normal thyroid tissue through a transverse incision in the creases of the neck. This operation leaves a scar which is almost invisible. Should this parenchymatous tumour cause pressure upon the trachea, it will be of course impossible to enucleate it, and we must therefore remove one lobe, tie the thyroid arteries and veins of that side, and divide the isthmus and remove one entire lobe—an operation which is called thyroidectomy.

This case is one of interest not only because of its rarity, but because it is a stumbling-block in the matter of diagnosis to the unwary practitioner and to the unfortunate candidate who encounters one of these cases at an examination. It is a case of *a supernumerary cervical rib.* In the posterior triangle of the left side of the neck of this woman you will perceive an obscure swelling, and on palpating it you will encounter a firm, hard, fixed mass which cannot be moved upon the skeleton. But the deceptive point about this lump is that it most obviously pulsates, and that if this pulsation is investigated from within out, it is found to be expansile. However, if you carefully palpate the tumour from before back, you will find that there is no expansile pulsation. These signs are easily understood when you remember that the subclavian artery almost invariably hooks over a supernumerary cervical rib, and it is the pulsating kink in the artery which is felt. These supernumerary ribs are doubtless congenital in origin, although they may perhaps increase in size as life goes on, but they are as a rule only discovered in later life, and usually when the patient has become thinner for some reason. In most cases they produce no symptom, and therefore as interesting and harmless peculiarities they should be left alone. But it nearly always happens that these patients ultimately encounter some enthusiastic surgeon, who gratuitously removes this interesting deformity. In a few cases they do appear to produce severe symptoms by involving the cords of the brachial plexus, but I have never encountered such a case.

This man has, I believe, *a neuroma of his musculo-spiral nerve.* On the outer side of his arm, about three inches above the external condyle, in the situation of the external intermuscular septum, you will find a round, smooth tumour the size of a large hazel-nut. This tumour moves from side to side, but not upwards and downwards. The lump, he says, has been there all his life. It is very tender on pressure, and produces shooting pains down the back of the forearm, apparently in the distribution of the radial nerve. When the man first came to my out-patients' he was complaining of loss of power in his grasp, and of numbness in the distribution of the radial nerve. One of the students who examined him apparently mistook the swelling for a ganglion, which it closely resembles, although, of course, there is no joint or tendon sheath in the neighbourhood. He endeavoured to burst the supposititious ganglion by pressure with his thumb, and although it caused the man violent pain, he attributes the disappearance of the numbness and of the loss of power to this mistaken treatment. To-day you will see that with the exception of the tumour he exhibits no signs. Under these circumstances I propose to leave the tumour alone, and not to risk the chance of the nerve failing to unite after resection. I will now show you *a case of coxa vara in a little girl of nine* years of age. Eighteen months before I first saw her she was found by her mother crying on the beach at Southend and complaining of a pain in her hip. She was unable to walk for three days, and after this she got about fairly well for a time, when progressive limp and disability in the hip supervened, so that after eighteen months her mother brought her to me quite lame. On examining this child you will see at once that the trochanter is very prominent on the right side, and that it is on the same level as the anterior superior spine of the ilium, whereas on the left side the trochanter is 1½ inch below the corresponding point. The movements of the hip appear to be quite free from pain, but they are limited in a way characteristic of coxa vara. If I fix the pelvis in a symmetrical position and abduct the two legs as far as possible, you will see that the right leg can only be abducted

Digitized by Google

about thirty degrees, whilst the left can be abducted nearly eighty degrees from the middle line. This limitation of abduction is typical. Inversion of the femur is also greatly limited. Both these signs are due to the bending of the neck of the femur in two directions. It is bent downwards, so that in abduction the great trochanter almost immediately strikes the edge of the acetabulum. It is bent with a convexity forwards, so that the anterior surface of the neck meets the edge of the acetabulum when we endeavour to invert the femur. When I measure the two legs from the anterior superior spine to the internal malleoli, you will see that the right measures only $19\frac{1}{4}$ inches, whilst the left is $20\frac{3}{4}$ inches. A shortening of $1\frac{1}{2}$ inches could not occur in tubercular disease of the hip-joint without dislocation of the head of the bone on to the dorsum of the ilium, and it is very clear that the head of the bone is in the acetabulum and working well in this case. The skiagraph (see Fig.) of this case shows very well not only the bending of the neck of the femur, but also the substance of the bone has been largely absorbed so that the neck is actually shorter. This case was shown at the Clinical Society of London last year, and it was suggested there that this child had had a small focus of tubercular osteomyelitis in the neck of the femur, and that this weakness, combined with some slight accident upon the beach at Southend, had led to a green-stick subperiosteal fracture of the neck of the femur. When the child continued to walk upon this bone before it had united, the neck gave way under the strain and became bent and partly absorbed. It is usual to speak of coxa vara as being divided into two classes, true and false. It is a common custom in surgical nomenclature to speak of the first variety of a disease which is described as the true one, and of all subsequent varieties, quite independently of their importance, as being false. This appears to be a clumsy and undesirable method, and particularly so in the case of coxa vara. If we take coxa vara to be simply a bending downwards and perhaps backwards of the neck of the femur, then



Digitized by Google

we may describe five varieties of it occurring at different ages. First, a child may be born with the neck of its femur so deformed, and this is the congenital variety. Secondly, in rickety children, the whole of whose skeleton is bending under their weight, it is not unusual to find that the neck of the femur has shared in these deformities, and this is known as rickety coxa vara. Thirdly, as Royal Whitman, of New York, has insisted, between the ages of four and eight the typical injury to the head and neck of the femur from trauma is a fracture through the neck, and this fracture may be either complete or subperiosteal. The history of such a case is very similar to that of the child I show you to-day; after a severe fall the child is unable to walk for three or four days. It then gets about for two or three months with more or less inconvenience. After this progressive disability, limping, growing out of the hip, and shortening supervene, and the child is found to be in a typical condition of coxa vara. These signs are of course due to the partly united neck of the femur bending under pressure. Fourthly, at the age of fourteen to eighteen boys frequently separate the upper epiphysis of their femora. The neck of the femur naturally slips up, and the epiphysis remaining in the acetablum becomes united to the lower part of the neck, thereby producing an almost horizontal neck to the femur. This is coxa vara subsequent to separated epiphysis of the femur. Fifthly, and finally, we have true idiopathic coxa vara which supervenes in rapidly growing youths about the age of seventeen or eighteen. Four elements appear to take part in the causation of this variety of coxa vara. In the first place the body-weight is rapidly increasing at the time of puberty, and this extra weight has to be borne by their skeletons. Secondly, this skeleton is rapidly growing and is forming soft new bones, and, indeed, in many cases may be said to be jerry-built. The increase in height in boys at this age is almost entirely produced by the rapid growth at the upper, but especially at the lower end of the femur. It is interesting to note that the two static deformities common at this age are situated one at the upper and one at the lower end of the femur, *i. e.* coxa vara and genu valgum. Moreover the muscles are atonic and do not take their proper share in supporting the body-weight, thus throwing an increased strain upon the softened bones. Lastly, it is at this age that the youth quits school, and as he is as yet unskilled in any trade he is put to do all the heavy manual work, particularly carrying heavy parcels and packages, which throw an additional strain upon the neck of his femur. It is not surprising that with all these coincident factors at work the neck of the femur should bend most commonly at this age. The treatment may be dismissed in a few words. In children a subcutaneous osteotomy of the neck of the femur may be performed by simply abducting the femur under an anæsthetic to a similar degree to the normal side, and then putting the child up in plaster. The neck of the femur is broken across the projecting rim of the acetabulum. In adults it is necessary to do a cuneiform subtrochanteric osteotomy, which will restore the obliquity of the femoral neck.

In this child I contented myself with the application of a Thomas' hip splint for a year, and then I ordered a boot which exactly compensated for the shortening. The mother of our little patient now tells me that she fears the leg is wasting away, because they have had to increase the length of the clump boot from 1½ inches to 2 inches. This is a very common delusion in the case of all shortened legs, and it leads very naturally to the pernicious practice of progressively increasing the height of the clump shoe until it may be five or six inches high. You see when I measure the legs once more there is but 1½ inches shortening on the right. The extra apparent shortening is due to the bad habit these patients all tend to acquire of tilting up their pelvis on the side of the weaker leg so as to throw more weight on the healthy one. If this pernicious habit is encouraged by ever-increasing length of boot the pelvis becomes tremendously tilted, spinal curves appear, and the patient is doomed to drag about with him for the rest of his life a huge clump boot. I therefore impress on this mother that this clump boot must not exceed 1½ inches until the measurement of the legs shows that the actual shortening is greater.

We have received a sample package of "antiphlogistine," for which substance it is claimed that it can be used with advantage as an external application in inflammatory conditions. It is not only an elegant preparation, but it is also an efficacious remedy always ready for immediate application as a soothing and antiseptic poultice and surgical dressing.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 585. WEDNESDAY, JANUARY 13, 1904. Vol. XXIII. No. 13.

CONTENTS.

PAGE

*The Value of Symptoms in Diagnosis. By JOHN ROSE BRADFORD, M.D., F.R.C.P., F.R.S. 193

*Cases Illustrating some Rarer Types of Pneumonia in Infants. By S. VERE PEARSON, M.B., M.R.C.P. 200

*Two Lectures on Piles, their Nature and Treatment. By J. JACKSON CLARKE, M.B., F.R.C.S. LECTURE II ... 206

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½, *Bartholomew Close, London, E.C.*

THE VALUE OF SYMPTOMS IN DIAGNOSIS.*

By JOHN ROSE BRADFORD, M.D., F.R.C.P., F.R.S.,

Physician to University College Hospital.

THE diagnosis of disease must always remain not only one of the most interesting, but also one of the most important problems in medicine, second in importance only to an accurate knowledge of the nature of the causes of disease. Measures directed to the prevention, to the cure, or to the relief of disease are often devised with comparative ease if our knowledge of the nature of disease and our methods of diagnosis are complete and thorough. Even in the case of many incurable diseases efficient measures can be devised for the restriction of their incidence. Thus, in the case of such a malady as plague, where the virus reaches the human body from a similar affection in animals, efficient means have been devised in many countries, and especially at ports, to restrict the distribution and the spread of this disease.

For the successful treatment of disease it is not only necessary to have an accurate knowledge of its cause and nature, but it is equally important that methods of diagnosis should be accurate and thorough. This is necessary for many reasons. In the first place it is essential, both in the treatment and in the prevention of disease, for the malady to be recognised both in its early stages and also in what may be called its mild varieties. No malady, perhaps, illustrates this better than tuberculosis. Everyone will admit that for the successful combating of this affection it is essential to recognise the very earliest forms of the disease. It is perhaps not so universally recognised that it is equally essential, if possible, to recognise the

* Delivered before the West Kent Medical Society on December 4th.

Digitized by Google

mild and the latent forms of the disease, that is to say, those instances of the malady where perhaps the general health of the individual is not very seriously compromised. This is important, not so much perhaps in the interests of the person affected as in those of the healthy. In the modern discussions on the prevention, or the possibility of prevention, of pulmonary phthisis, the fact is sometimes overlooked that very large numbers of apparently healthy persons have tuberculous lesions of varying degrees of development present in their bodies, and that these lesions, if not a source of great danger to their possessors, are at any rate potential causes of the spread of the disease to others.

Again, a large number of the infective, and certainly the acute specific diseases, are undoubtedly propagated by slight and mild cases of the malady. This is notoriously the case, for instance, in smallpox and in enteric fever, and perhaps in dysentery. In all these affections the malady may exist in so mild a form as to attract but little notice, and yet be capable of causing wide dissemination under suitable circumstances.

The accurate diagnosis of disease is essential for its correct treatment for another reason. It not uncommonly happens that under a given clinical description a variety of separate pathological processes is really included. This is the case, for instance, in such a malady as malignant or ulcerative endocarditis, which is now known to be dependent on a great variety of causes possessing but little in common with one another. The same remark is also probably true with reference to diabetes mellitus; the ordinary symptoms associated with diabetes are probably not characteristic of a single morbid process dependent on a single cause, but we have rather to deal in this condition with a particular group of symptoms possessing a general resemblance in different cases, but really dependent on or associated with different morbid processes. An accurate differential diagnosis of the different varieties of such affections is of the greatest importance as regards their successful treatment, and the absence of this has not uncommonly led to the discrediting of methods of treatment which are really very valuable in suitable cases. An instance illustrating this is afforded by the use of antistreptococcic serum in the treatment of malignant endocarditis. Some observers have obtained very good results, others have seen little or no beneficial effect, and thus the remedy is frequently discredited; whereas the real cause in the differences of the results obtained is very probably to be associated with differences in the nature of the infection. In the one case the endocarditis is associated with a streptococcal infection, and in other cases with some other micro-organism. A fact of the same order is seen in the dietetic treatment of diabetes, successful in some cases, unsuccessful in others, the differences depending really not on chance, but on the fact that they are different forms of the disease probably associated with different lesions, and possibly dependent on different causes; and the time is coming when it will not be sufficient to diagnose malignant endocarditis or diabetes, but it will be necessary to make a more accurate and complete diagnosis as regards the particular form of infection or the particular morbid process present.

Although we are all in the habit of emphasizing the importance of accurate diagnosis, yet I doubt whether we always realise the very far-reaching power that really accurate diagnosis gives one in the practice of one's profession. The great majority of the many difficulties and uncertainties in the practice of medicine arise from imperfect or inaccurate diagnosis, very often dependent on the present imperfect stage of our knowledge of medicine, but in some instances really dependent on our want of appreciation of the signs and symptoms presented by disease.

It is customary to consider the diagnosis of any given malady as deduced from the study of three different sets of facts: those elicited from the patient or from others as regards the mode of onset, etc., and these are included under the term of the history of the patient; secondly, those deduced from the patient's statements as to the particular discomfort or other effects produced by his illness, which we talk of as symptoms; thirdly, those deduced from the observation of the physician himself, and which are spoken of as physical signs. All these three separate series of effects have their value, and in different maladies are of different relative importance. The first two have, of course, the obvious drawback that they are liable to be somewhat biassed statements, consciously or unconsciously. In the case of the third there is the somewhat grim satisfaction that any errors made

Digitized by Google

are usually errors of our own, for which the patient is not responsible.

For the last fifty years the attention of practitioners has been mainly concentrated on the study and development of the diagnosis of disease by physical signs, owing to the precision of many of the methods that have been introduced and the very great value of the results achieved. It is only necessary to quote the thermometer, the stethoscope, the ophthalmoscope, the chemical methods for investigating the excreta, the discharges, and the secretions, to show to what a great pitch of excellence the methods of physical examination have reached. Men have become so impressed with the value of physical signs that it is not uncommon for the diagnosis to be made entirely by physical signs, and for the history and the symptoms to be almost neglected. Physical signs as a rule only give information as regards the anatomical seat of the lesion; they do not give information necessarily as regards the pathological nature of the lesion. Many of the errors of diagnosis in disease are made from confusing these two totally distinct phenomena. Physical signs will enable a physician to diagnose the presence of a lesion which may be, for instance, in the internal capsule of the brain or in the mitral orifice of the heart; and very often these methods are of such great exactness as to render such diagnosis extremely accurate. In some instances the diagnosis of the anatomical seat of the lesion—as, for example, at the mitral orifice—practically diagnoses the nature of the lesion, inasmuch as the number of affections of such a structure as the mitral orifice are very limited, and their nature not very varied. In such an instance as the internal capsule, on the other hand, the number of possible lesions in such a situation is very considerable, and really the whole future of the patient may depend more on the accurate recognition of the nature of the lesion than on the accurate recognition of the seat of the lesion; and physical signs may absolutely fail to give accurate information as regards the nature of the morbid process.

Accuracy in the detection and interpretation of physical signs requires long experience, but on the whole the correct interpretation of symptoms, and still more, perhaps, the eliciting of symptoms, is a more difficult art. In order to elicit symptoms it is necessary to cross-examine a patient somewhat after the legal style, and especially to adhere to the rule of not asking leading questions, as most patients seem to be anxious to answer a question in the sense that they imagine the doctor wishes it answered.

The study of symptoms is a study to which sufficient attention is often not directed during the practitioner's career as a student, and my main object in addressing you on the value of symptoms in diagnosis is my belief that at the present time this is neglected owing to the very great development and importance attached to physical signs.

In order to elicit a history of symptoms satisfactorily, it is often no doubt necessary to have a suspicion of the nature of the patient's illness, and this after all can often be obtained not only from the statements of the patient, but also from the general appearance and facies. The great importance of the thorough study of symptoms must be at once granted when we remember that in the vast number of functional diseases symptoms only are present; and further, as we shall see later, symptoms only may be present, or at any rate obtrusive, in cases of far advanced organic disease.

In a general consideration of the value of symptoms in diagnosis we may classify our subject roughly, as follows:

1. In functional diseases symptoms only may be present.

2. Gross organic diseases may have advanced to a very high degree of development without producing any very obvious physical signs; and although it is true that in some instances symptoms may also be absent, so that the malady may be truly described as running a latent course, yet in a number of instances of gross organic disease careful inquiry will elicit the occurrence of characteristic symptoms.

3. In some organic diseases symptoms only are present.

4. The onset of complications in the course of the progress of organic disease is heralded by symptoms, and frequently if we wait for physical signs the opportunity for successful treatment has by then passed away.

5. In many instances the occurrence of characteristic symptoms affords the clue to the real nature of organic diseases, and is especially valuable in determining the actual pathological nature rather than the seat of the lesion.

Digitized by Google

6. Lastly, though foreign to our subject of this evening, the occurrence of symptoms in the course of organic diseases may often be the best indication that we have for treatment.

1. *Functional diseases.*—A very large number of the affections we have to deal with in practice fall under the category of functional diseases, that is to say, maladies the exact nature of which is not understood, and the underlying lesions are at present often unknown. In many functional diseases no doubt some physical signs may be present, but in others the diagnosis has to be made entirely on the nature of the symptoms, associated, perhaps, with the absence of physical signs indicative of any organic disease. It would be wearisome to enumerate all the neurotic and neurasthenic affections where symptoms only are present. In many of these the very remarkable character of the subjective sensations that the patient complains of affords a ready clue to the differentiation of the malady from organic diseases, with which at first sight they might be confounded. A convenient illustration is afforded by the really extraordinary subjective sensations in the head that neurasthenic patients complain of. They will almost invariably at first state that they suffer from headache, but cross-examination very soon reveals that it is not a headache in the strict sense of the word, but a distressing sensation of very varied character, such as opening and shutting of the head, a weight on the head, a crawling sensation, a burning sensation, etc. The most characteristic of these, perhaps, is the sense of intolerable weight on the occipital region of the head and on the nape of the neck, situations in which pain from organic disease is at any rate not common, although in cervical pachymeningitis pain of a somewhat similar distribution is sometimes seen from implication of the posterior roots. The elaborate character of the sensations is very often the main indication as to the neurotic or neurasthenic nature of the affections, whereas if the patient's statement of the headache be too readily accepted, serious errors of diagnosis may be made.

One of the most important functional diseases of the nervous system where the diagnosis has to be made, in the vast majority of cases, entirely on the symptoms, is epilepsy, and more especially the anomalous forms of epilepsy where convulsions are absent, and especially in "petit mal."

Petit mal presents itself under a great variety of forms, and it probably is not an exaggerated statement to say that a great number of the cases are not really correctly diagnosed, owing to a wrong interpretation being placed on the statements of the patient, or to the symptoms not being elicited in the course of our examination. Many such cases are looked upon not only by the sufferers, but also by their medical advisers, as fainting fits; whereas this mistake would be avoided if due stress were laid upon the significance of such symptoms as numbness and tingling in some part of the body, or the strange visceral discomfort in the form of a distressing sensation rising up from the abdomen to the chest, and then followed by some blurring or temporary obscuration of consciousness. Palpitation is also a symptom which ought not to be too readily attributed to primary cardiac or vaso-motor disturbance, for it is really often of central and epileptic origin. In a great number of cases of petit mal careful interrogation will elicit a history very strongly suggestive of the presence of an epileptic aura of some kind, and will at once remove the case from the category of so-called fainting. In the great majority of cases of epilepsy itself we are not present at the time of a fit, and have to form our opinion as to the nature of the seizure entirely on the history and on the symptoms. It is often very difficult to obtain a history that is satisfactory from a patient's relatives or friends, and the convincing evidence that the seizure is really due to epilepsy and is not hysterical is obtained by a careful eliciting of some of the more characteristic symptoms of epilepsy, and more especially, perhaps, of the aura.

Ménière's disease, again, is one which very often has to be diagnosed on the strength of the symptoms rather than on any results of physical examination.

2. *Gross organic diseases.*—Some of the most serious gross organic diseases may reach a very high stage of development and yet produce few, if any, physical signs, and only reveal their presence by the occurrence of symptoms which, in some of the more fatal of them, may be misinterpreted. Many instances of this might be quoted, but it will suffice to mention two or three. Hepatic cirrhosis may reach a very high degree of development, and yet symptoms only may be present, and not uncommonly referred to the stomach rather than to

Digitized by Google

the liver. The same is true of such a common disease as gastric ulcer, and it would not be too much to say that in the majority of cases the presence of this malady is to be diagnosed rather by an intelligent appreciation of symptoms than by the occurrence of physical signs. The peculiar situation of the pain, as well as its relation to food, affords valuable indications, and it is even possible that the character of the pain may give some information on even such a point as the depth of the ulcer. Thus continuous pain in this affection may be dependent on the ulcer being sufficiently deep to have led to changes in the peritoneum and the production of adhesions.

Hæmatemesis is perhaps not always correctly described as a symptom, but whether this be so or not it is of some importance to bear in mind that it may occur, and even be fatal, without the presence of what is ordinarily spoken of as the typical gastric ulcer.

Diseases of the cerebral arteries afford a very good illustration of the occurrence of very serious and even fatal organic disease which may produce few, if any, physical signs, and only reveal its presence by such a symptom as intense headache. It is true that in cases of this kind a diagnosis cannot be made in many instances, but that does not diminish the importance of recognising that this very serious malady is a cause of intense headache; and a shrewd suspicion can sometimes be formed that thickening of the cerebral vessels or an aneurysm of one of them may be the cause of severe headache, in the absence of physical signs, such as optic neuritis, etc., pointing to the presence of cerebral tumour. Many instances are on record where the rupture of a cerebral aneurysm or the blocking of a large cerebral artery has occurred in a patient who only presented symptoms such as severe headache for a variable period before the onset of the fatal result.

Another malady in which symptoms only are present for a considerable time, and where diagnosis may not uncommonly be hazarded on their presence alone, is Addison's disease, where palpitation, cardiac weakness, pain, and vomiting may be the only phenomena produced for weeks or months; and cases have been recorded where the characteristic pigmentation (which, perhaps, must be present with the symptoms in order to justify the diagnosis) has only occurred quite late in the history of the disease. It is clearly a matter of great importance to recognise such a malady as Addison's disease, and not to confuse it with some trivial ailment, as may be readily done if we lay too much stress on the presence or absence of physical signs. The occurrence of the symptoms may not perhaps justify a diagnosis, but at any rate they may justify a suspicion being entertained of the existence of grave organic disease.

3. *Organic diseases in which symptoms only are present.*—Renal and biliary colic afford good instances of organic diseases in which symptoms only may be present, and where the differential diagnosis has often to be made almost entirely on the symptoms. Doubtless in some instances enlargement or tenderness of the kidney or evidence of local changes in the gall-bladder may be elicited, but in a very considerable number of cases of renal and biliary colic such physical signs are absent, and the differential diagnosis has to be made entirely on the situation of the pain. In cases of biliary colic this applies especially to those cases where the calculi are small and merely produce symptoms in their passage along the cystic duct. The distribution of renal pain, that is to say, the situations in which it is felt, is quite distinct from the area in which the pain of biliary colic occurs. The difficulty that presents itself not uncommonly in the diagnosis of biliary colic is to differentiate the pain produced by this disease from that occurring in various gastric affections; and perhaps, on the whole, the mistake most commonly made is to attribute a pain in the right hypochondrium to hepatic causes when it is really of gastric origin, as it is difficult for us to remember always that the stomach is a mesial and bilateral organ, and not really a left-sided one.

4. *Onset of complications.*—It may perhaps be said that one of the most important subjects connected with the study of symptoms is the information they afford of the onset of complications in gross organic disease. In many instances doubtless these complications are necessarily fatal, but in others the only chance of saving a patient depends on the prompt recognition of the nature and cause of these symptoms. Many illustrations of this might be quoted, but it will suffice to mention the occurrence of perforation in enteric fever, in appendicitis, in gastric ulcer, or in obstruction of the large intestine, or the onset of uræmia in renal

Digitized by Google

disease, or of coma in diabetes. In enteric fever, in gastric ulcer, in appendicitis, and in uræmia everything may depend on the prompt recognition that the new symptoms, occurring in the course of the patient's illness, are really to be attributed to the complications mentioned. In cases of perforation, if treatment is delayed until all the characteristic physical signs of the presence of gas in large amount in the peritoneum are present, the chances of successful interference are very greatly diminished. The diagnosis must often be hazarded on the mere situation and character of an attack of pain, sudden in its onset, or perhaps of such a symptom as the sudden occurrence of vomiting not due to any ordinary cause. In such diseases as kidney disease and diabetes the onset of uræmia or of diabetic coma is necessarily shown rather by the occurrence of symptoms than of physical signs, and it is not always that a slight increase in or sudden occurrence of breathlessness, irritability, or in diabetes of abdominal pain, is correctly interpreted as really the initial manifestation of a serious toxic state. The only chance of the successful treatment of diabetic coma lies in its recognition at the earliest possible moment, and even then it is not as hopeful as the treatment of uræmia.

5. *Symptoms as a clue to diagnosis of the nature of the lesion.*—One of the best illustrations of the value of symptoms in diagnosing the nature of a lesion is perhaps the common occurrence of vomiting in cases of cerebral hæmorrhage. The sudden occurrence of hemiplegia almost always raises the question as to whether the lesion is thrombotic, embolic, or due to hæmorrhage. Doubtless in many instances this question can be answered from the physical signs, or inferentially by the age, past history, etc., or accompanying morbid processes in other organs of the patient. In a great many cases the history of the symptoms at the onset, and especially, perhaps, the occurrence of vomiting, affords conclusive evidence of the presence of cerebral hæmorrhage.

This frequent occurrence of vomiting in cerebral hæmorrhage is not only of importance in diagnosing the hæmorrhage, but is of still greater importance as a symptom of a grave cerebral lesion, as many attacks of cerebral hæmorrhage have been erroneously imputed to merely trivial gastric trouble, and their serious nature quite overlooked. This has occurred even in cases where paralytic lesions have subsequently become developed, and is necessarily still more apt to occur if the hæmorrhage should happen to take place in a region of the brain, destruction of which is not followed by motor paresis. Since cerebral hæmorrhage may be ushered in suddenly with severe vomiting, together with a certain amount of collapse, and only transient weakness be present, the whole nature of the illness may be entirely misunderstood, and attributed to some trifling indiscretion in diet, and a most erroneous diagnosis and prognosis made.

Another symptom often throwing unexpected light in diagnosis is the numbness and tingling of the extremities so characteristic of neuritis, and occurring as a very early symptom in this disorder. Further, the pains which occur in neuritis are very apt to be referred to or felt in the vicinity of joints, and hence are attributed by patients to rheumatism, but the true nature of such pains may often be surmised from the accompanying numbness and tingling in the extremities.

Pain is unquestionably the symptom which gives the most information in diagnosis, but it is often difficult to interpret correctly unless care be taken to differentiate carefully between referred pains and genuine local pains; and in order to correctly interpret both varieties of pain it is very necessary to possess an accurate knowledge of the distribution not only of cutaneous nerves, but more especially the relation of the posterior roots of spinal nerves to the surface of the body, as worked out by Sherrington in animals and Head in man. Many root pains, more especially, perhaps, those of tabes dorsalis, may be felt in the vicinity of joints, and attributed to joint trouble when there is no such mischief present, the erroneous interpretation depending on the fact that the cutaneous distribution of certain posterior roots may include areas in the vicinity of certain joints; and it would seem as if pain were constantly referred to the junction of two cutaneous areas.

The question of referred pain in visceral disease is much too large a one to enter upon this evening, and I will only allude to the very accurate diagnosis that may sometimes be made by careful attention to the area in which pain is felt in certain visceral diseases.

Everyone is familiar with the characteristic distribution of the pain of angina pectoris and its

Digitized by Google

relation to serious organic disease of the heart and great vessels. So much so is this the case that in all cases of pain in the arm the suspicion of angina is entertained whenever the pain involves the area on the inner side of the arm so characteristic of this affection. When we consider the frequency with which pain down the arm is associated with thoracic disease, and the generally correct interpretation that is given to such pain, it is rather remarkable that pain down the leg should be so frequently attributed to lesions in the leg itself rather than to its probable cause, viz. abdominal and pelvic disease. As just mentioned, in all cases of pain down the arm the suspicion of angina probably crosses the mind of the physician, whereas in pain down the leg a neuritis, sciatic or other, is almost always the first suspicion entertained. As a matter of fact, a large number of abdominal and pelvic diseases reveal themselves in the first instance by causing pain down the leg, which not uncommonly is looked upon as sciatica. Doubtless a great many cases of so-called sciatica are associated with chronic affections of the hip, just as many cases of pain down the arm attributed to brachial neuritis are really associated with similar chronic disease in the shoulder-joint. There can, however, be but little doubt that a great many cases of so-called sciatica are really instances of referred pain associated with prostatic, vesical, uterine, rectal, and even renal diseases.

Another instance of referred pain that must often have afforded the greatest assistance in diagnosis is the pain that the top of the shoulder and the lower part of the neck present in a number of affections in which the diaphragm, either on its pleural or peritoneal aspect, is inflamed. Such pain is seen in pericarditis, pleurisy, pneumonia, subphrenic abscess, peritonitis, hepatic abscess, and a number of diseases which are often extremely obscure. The occurrence of this pain, characteristic in its area, may be the first symptom suggesting the presence of general peritonitis; thus perforation of the stomach may occur, causing at its onset severe pain in the abdomen, which perhaps passes off, and the general condition of the patient may be absolutely misleading and not suggest the presence of the grave complication of peritonitis. The presence of this pain may literally be the first symptom pointing to the conclusion that the peritoneum on the under surface of the diaphragm is inflamed. Similarly it may afford valuable evidence of the presence of such a condition as a diaphragmatic empyema, and it is quite common to see this symptom at the onset of pericarditis. I have known of a case of pericarditis where the erroneous diagnosis of double aortic disease and rheumatism of the left shoulder was made, owing to the to-and-fro friction being interpreted as a double murmur, and the pain in the shoulder attributed to rheumatic affection of the joint.

There is scarcely any symptom presented by disease which one might not take as illustrating the importance of the study of symptoms, and it will suffice if only two or three are mentioned. Thus delirium is not only a well-known accompaniment of pyrexial processes, but it may also afford valuable information of the presence of other morbid processes which may, perhaps, owing to a variety of circumstances, not have declared themselves fully by physical signs. For instance, a patient suffering from violent delirium may really be affected with pneumonia; or again, it may be an indication of the presence of pericarditis, or even of myocarditis; and, as is well known, it is by no means an unusual accompaniment of various chronic lesions of the heart, more especially, perhaps, aortic regurgitation.

Dyspnœa, again, in its very varied forms will often be of great assistance. Thus the hissing dyspnœa of uræmia is extremely characteristic of the condition, and may be really the only symptom pointing to the very grave condition of the patient. The clinical significance of Cheyne-Stokes breathing is well known, pointing often to the presence of curious and obscure degeneration of the heart which does not reveal itself by ordinary physical signs. The great breathlessness of acetonæmia is also an extremely characteristic symptom, and one of great value in the recognition of the nature of sudden illness in a patient seen for the first time.

A remarkable form of dyspnœa, also characteristic but not very frequently seen, is the urgent, panting respiration seen in exophthalmic goitre, and frequently of fatal import.

In some organic diseases, like Bright's disease and diabetes, the sudden occurrence of sleeplessness may be a symptom of the very greatest importance, marking as it does the onset of uræmia or acetonæmia, as the case may be.

Digitized by Google

The occurrence of hiccough in abdominal diseases, especially peritonitis and in enteric fever, may often be a symptom of great practical importance.

These afford sufficient illustrations of the value of the study of symptoms even in gross organic diseases.

As mentioned above, pain is undoubtedly the most valuable, and one where with a thorough knowledge of anatomy great assistance may be afforded to diagnosis. The most striking instance of this, apart from the occurrence of referred pain in visceral diseases, is afforded by the root pains present in a number of diseases of the spinal membranes or of the vertebræ, and every practitioner is aware of the possibility of mistaking such an affection as caries of the spine with some trivial gastric disorder, unless by the careful interrogation of the patient he assures himself that the pain has the characteristic root distribution.

Very curious associations are sometimes seen in affections of the spinal membranes or of the bones. Thus I have seen a patient who came complaining first of loss of power in the arm associated with headache, and where for the moment the suspicion of cerebral tumour was obtained. But on cross-examination it became clear that pachymeningitis in the cervical region was the cause of the pain in the neck, which passed up to the occiput, and the weakness of the arm was dependent on pressure on the anterior roots, just as the pain present was due to similar damage to the posterior roots.

The fact that any given pain experienced by a patient is really a root pain can generally be elicited by the characteristics of the distribution of such pains.

6. Lastly, *the occurrence of symptoms may afford valuable indication for treatment.* It is difficult sometimes to diagnose, for instance, the amount of fluid present in a pleural effusion. The occurrence of dyspnœa with pleural effusion will always be an indication for paracentesis, and in reference to this it should never be forgotten that dyspnœa in chronic cardiac disease is often associated with the rapid development of a pleural effusion running a latent course.

In many of the diseases quoted this evening the occurrence of sudden symptoms is not only the means of making the diagnosis, but they also at the same time give indications for valuable treatment. Notable instances of this are the onset of perforation in enteric fever on the one hand, and the onset of uræmia in renal disease on the other hand.

Thus, whether we look at medicine from the point of view of diagnosis or prognosis or treatment, in all cases it is most important to pay great attention to the study of symptoms if success is to be achieved.

CASES ILLUSTRATING SOME RARER TYPES OF PNEUMONIA IN INFANTS.

By S. VERE PEARSON, M.B.Cantab., M.R.C.P.Lond.,

Assistant Physician, East London Hospital for Children, Shadwell; Medical Registrar, St. George's Hospital.

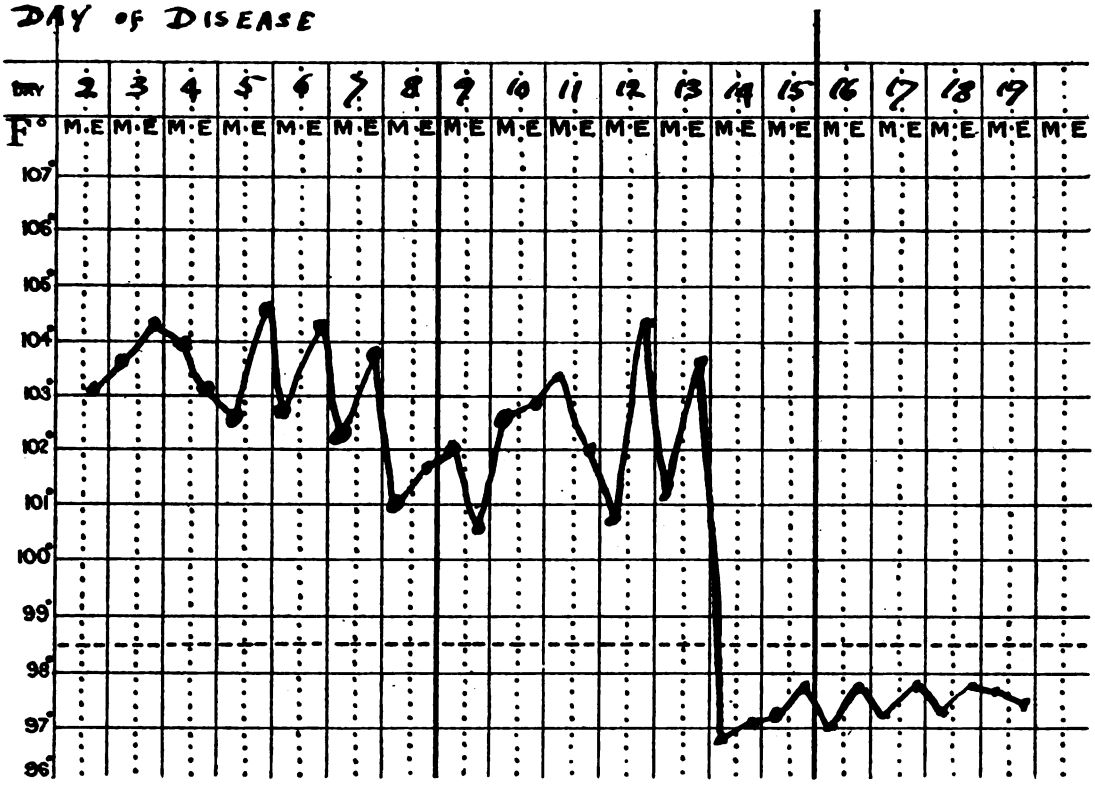
THE following cases are interesting, as they exemplify certain unusual symptoms of pneumonia in infant patients. They illustrate, too, some of the difficulties which may arise in the diagnosis of pneumonia in infancy; and they show how mistakes in diagnosis and prognosis may arise, especially if too much attention is paid to the most obvious features and these are allowed to obtrude and to outweigh what appear to be minor symptoms. Certain well-recognised rules guide us in arriving at a correct diagnosis and in forming an accurate prognosis in cases of this kind. But in medicine every rule has its exception; some of the exceptions to the rules with respect to infantile pneumonia are exemplified below.

One of the most constant and reliable rules to guide us in the differential diagnosis between croupous and catarrhal pneumonia in infancy—a differentiation important on account of the much better prognosis of the former—is that a pneumonia which is primary and limited to the right upper lobe is a croupous pneumonia. If a primary pneumonia with signs limited to this region arises in a baby previously healthy, and the patient exhibits a simple tachypnœa with but slight prostration and no blueness or recession of the chest walls, it is practically certain to be croupous pneumonia. The first case which I am about to relate led me astray on account of the presence of most of these features.

1. The patient was a fine baby of 7 months who had been taken suddenly ill two days before I saw him. On examination there were well-marked signs of consolidation (dulness, tubular breathing, and bronchophony) limited to the region of the right upper lobe. He was breathing at the rate of 92 respirations per minute, but in spite of this sat up and was inclined to play with my stethoscope. There was, however, a little duskiness about the lips, and slight recession. The temperature was

Digitized by Google

103·6° In consideration of the fact that here was a previously healthy baby with an illness starting suddenly, and very soon giving rise to consolidation limited to the right upper lobe with but little prostration, I diagnosed croupous pneumonia and gave a good prognosis, explaining the slight cyanosis and recession by putting it down to a little obstruction in the infant's narrow upper respiratory passages, which are so easily constricted by a little reflex spasm or slight catarrh.



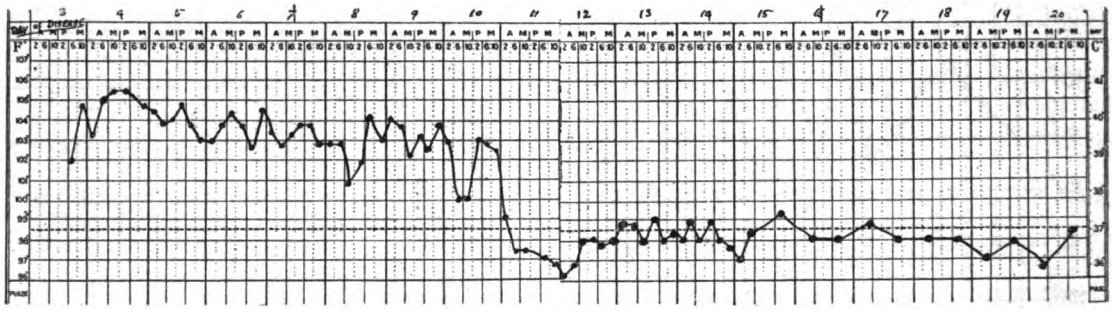
CASE 2.—Right apical croupous pneumonia with cerebral type of onset.

The child grew rapidly worse, cyanosis increased, laboured breathing and prostration soon made their appearance, and severe convulsions came on. The next time I saw the patient was forty-eight hours later, on the post-mortem table. The necropsy revealed catarrhal pneumonia with complete collapse of the right upper lobe. On considering the case afterwards I remembered that I had examined the patient in a poor light, so that probably there was more blueness at the time of my examination than I had recognised, and that I had heard an adventitious sound on the left side of the chest; but in view of the symptoms pointing, to my mind, so strongly in favour of croupous pneumonia, I had believed this to be accidental. I should have examined the patient in a better light, I should have taken more notice of the cyanosis and recession that were present, and I should have examined the left chest more carefully for râles; such were my conclusions after the event.

2. The second case presented difficulties in diagnosis of a different nature. It was a baby of one year old whose pneumonia was of the "cerebral" type. Text-books lay emphasis upon the fact that pneumonia in infancy may start with symptoms suggesting meningitis. This form of pneumonia is by no means common; it occurs in only about five per cent. of all cases. It is nearly always the croupous form of pneumonia that begins in this way. This patient's illness started rather suddenly. For several days general irritability, refusal of food, high fever, furred tongue, head retraction, and vomiting were the main symptoms, and no abnormal

Digitized by Google

physical signs could be discovered in the chest. Such a state of affairs in a baby naturally suggested meningitis; but pneumonia seemed to be the diagnosis most probably correct, for although there was great irritability with some hyperæsthesia, and although he showed much dislike to being disturbed, the mental state of the patient seemed to be quite clear. After a little experience with babies of this age, it is often possible to tell the difference between an irrational, purposeless, subconscious irritability very prone to arise in the case of an infant with meningitis, and the resentment to any interference often shown by an infant with pneumonia. The baby cried frequently; but the cry, though not unlike the piercing, distressful, short cry of meningitis, had not exactly those qualities. There were no other definite signs of meningitis, such as squint, dilated and sluggish pupils, irregular, slow, or variable pulse, muscular twitchings, or optic neuritis, some of which one would have expected to find after several days of such severe illness and fever as he had suffered. The breathing, too, was generally rather hurried, and never sighing or irregular; and the abdomen was a little distended rather than retracted. Then against meningitis there was the lack of conformity in the course of the disease to any of the various forms of meningitis. On the supposition that the patient was suffering from meningitis, it was difficult to fit in the symptoms with either purulent, tuberculous, or post-basic meningitis. The degree and persistence of the fever ought to have indicated septic meningitis. Yet there was no history of otitis media or of pneumonia, the two commonest immediately preceding causes of purulent meningitis, and the disease did not rapidly culminate in more definite signs of meningeal inflammation and death. Then against tuberculous meningitis was the absence of any wasting or anæmia, and the absence of history pointing to any prodromal symptoms, neither infallible tests, but both commonly present; also, usually, when a tuberculous meningitis does give rise to such a high temperature as was present in this case, which is not very often, the signs are as a rule advanced and well defined, and the patient's end very near. The acute nature of the illness, too, and the fact that the head retraction was not very marked, were against chronic basilar meningitis. For these reasons, then, the diagnosis of croupous pneumonia was made. On the seventh day signs in the lung appeared and proved the correctness of the diagnosis. The consolidation was of the right upper lobe; the temperature ended critically, though not until the fourteenth day, so that the disease ran a somewhat unusually long course. Delay in the appearance of physical signs occurs rather more frequently with croupous pneumonia at the right apex than with acute pneumonia affecting other regions. A rapid and perfect recovery ensued.

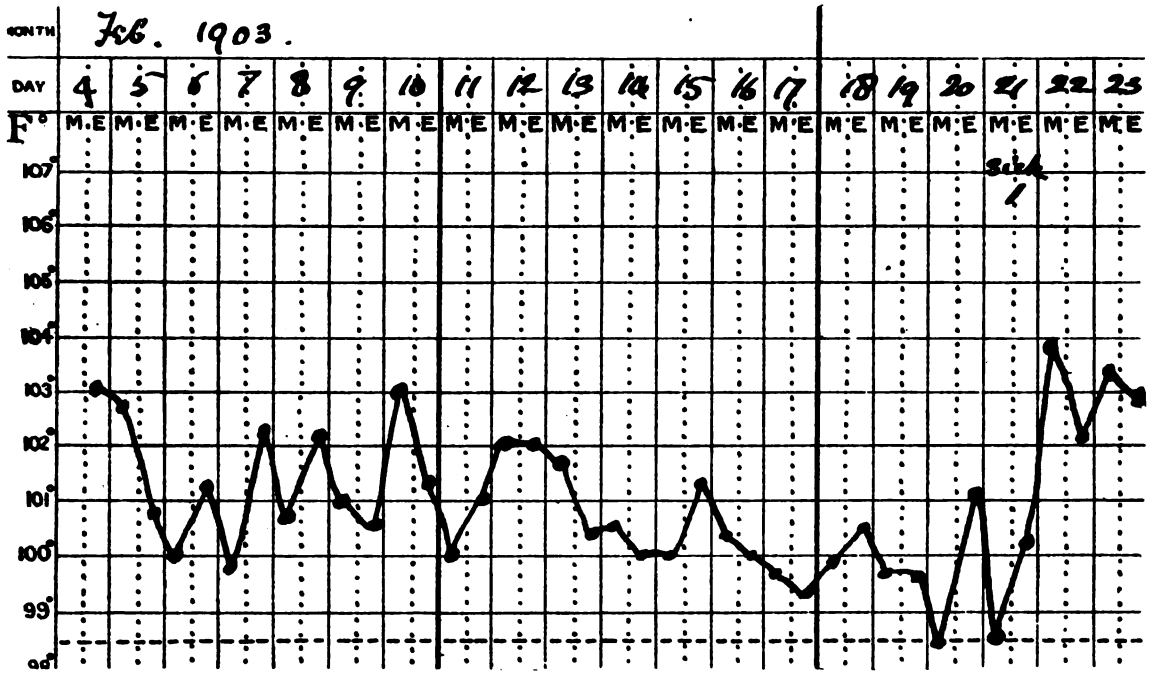


Case 3.—Primary broncho-pneumonia with critical ending to continuous pyrexia.

3. The third case is an example of a broncho-pneumonia of great severity, with high continuous pyrexia ending by crisis. It is usual for catarrhal pneumonia to have a remittent or intermittent type of pyrexia ending by lysis, and for the fever in croupous pneumonia to be more or less continuous, and to end by crisis. Also the duration of the pyrexia is longer in the former than in the latter, the average length of the one being often about three weeks against one week in the case of croupous pneumonia. The occurrence of continuous pyrexia is commoner in those broncho-pneumonias which arise primarily than in those

Digitized by Google

which succeed one of the exanthemata or bronchitis. The primary broncho-pneumonias have a better prognosis than the secondary; but usually those broncho-pneumonias, even though primary, which have a temperature of above 105° have a very grave prognosis. This is so especially if the child is under two years of age, for the majority of such cases die. This child to begin with was vigorous; she was 2¾ years old. She almost died, being for several days *in extremis*; but being originally vigorous and nearly three years of age, with the aid of free stimulation (strychnine and alcohol administered in abundance), she eventually pulled through and made a complete recovery. She may be cited as an example of the difficulties of prognosis, for for many days her death seemed imminent, and as an example of the happy results of careful nursing and medical supervision. In the matter of diagnosis this case had, especially in view of the type of pyrexia, to be differentiated from croupous pneumonia. The temperature chart is like that of croupous pneumonia, and this disease is sometimes complicated by bronchitis. The diagnosis of broncho-pneumonia, however, was easily established on account of the following features:— From the very start there were signs of disease in the lungs. For the first two days after admission into the hospital, *i. e.* on the third and fourth day of the disease, these were those of general catarrh of the bronchial tubes of medium and rather small size, with some impairment of resonance, and sharp smaller consonating râles at the left base. These signs of consolidation spread so that two days later consonating râles were heard in the left axilla and higher up the left back. At the same time the bronchial catarrh became more severe, and the general condition of the patient very soon became extremely grave. There was profound prostration, with much cyanosis and recession of the chest walls. A few fine metallic râles were heard now amongst the general catarrhal sounds on the right side, indicating some small scattered areas of consolidation on that side also. The child became pallid, dusky, and collapsed, and all this on the eighth day of the disease, when there was still no tubular breathing or bronchophony. Now it is the rule in croupous pneumonia to get a different state of affairs. Usually signs of consolidation limited to one lobe become definite quite early in the disease, and by the eighth day well-marked tubular breathing and bronchophony have been present for several days; this is especially so when abnormal signs in the lungs have made their appearance very soon after the onset of the disease. In this case,

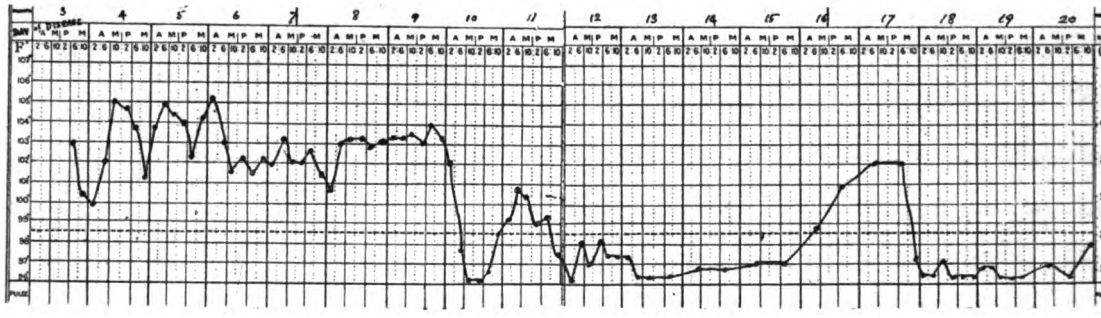


Case 4.—Acute broncho-pneumonia with pulmonary abscess formation, jaundice, and vegetative endocarditis.

Digitized by Google

on the other hand, it was not until the tenth day that well-marked tubular breath-sounds, with bronchophony, showed themselves. Again, if severe collapse and cyanosis have arisen in croupous pneumonia, there are usually extensive signs of massive consolidation; but in broncho-pneumonia, as in this case, grave prostration and blueness may arise without signs of massive consolidation. In tracing the further course of the case, too, we notice that the crisis was not, as is the rule in croupous pneumonia in well-nourished babies, very soon followed by an amelioration of the symptoms of collapse; for for several days after the crisis the grave prostration remained, and the patient's life hung in the balance. Moreover, instead of the signs rapidly undergoing resolution as soon as the fever ceased, as would most probably have occurred had the disease been cyanosis and recession, the slow resolution, and the tardy convalescence of the patient.

4. Occasionally broncho-pneumonia is but a sign of a general blood infection. The combination of broncho-pneumonia with septicæmia happens in all probability in three ways, or at all events such a supposition explains three different clinical types of the condition. The incidence of the infection falls upon the lungs at the same time that it affects other parts of the system. In the next type the lungs are invaded first, and secondarily to this general blood infection takes place; or possibly sometimes the invasion of pulmonary substance and blood is simultaneous, but the manifestations of lung involvement make their appearance before those of blood infection. In the third place the broncho-pneumonia is but a terminal event of the general micro-organic in-



Case 5.—Primary broncho-pneumonia and tetany ending fatally.

croupous pneumonia, the lungs took a long time to clear. On the thirteenth day of the disease there were still present dulness, tubular breathing, and bronchophony at the left base, and innumerable rhonchi and râles all over the rest of both lungs. There was still some dulness on the twenty-third day, and scattered adventitious sounds. The signs had only just disappeared by the twenty-eighth day from the onset. Some vomiting and diarrhœa complicated the case. Great weakness and anæmia lasted for a long time, but eventually the recovery was complete. In spite, then, of the sudden onset, the high continuous fever, and its critical ending, the disease was broncho-pneumonia and not croupous. To recapitulate, the chief points on which this diagnosis is based are these:—The scattered nature of the consolidations, the many catarrhal sounds, the late appearance of massive consolidation, the severe prostration, the marked vasion. Perhaps the last is the commonest event, but the association of septicæmia with broncho-pneumonia in any variety occurs infrequently; at least, it is rare as far as clinical evidence goes. From recent bacteriological investigation it would seem in reality to be a far commoner event than is generally supposed. The following case is a well-marked illustration of the second variety:

A baby of nine months was admitted into St. George's Hospital within a few days of the onset of his illness. He was well nourished, but had high fever (103°) and signs of catarrh of conjunctivæ and bronchi. He breathed rapidly, but was not much distressed. Five days later he was much worse; some cyanosis, inspiratory embarrassment, and signs of scattered consolidations in the lungs were evident, and left otorrhœa began. At the end of a week slight jaundice made its appear-

Digitized by Google

ance. A few days later, by which time massive consolidation had occurred over the upper half of the right lung, bronchial breathing and bronchophony being heard over this area, the jaundice had become much intensified, and there was double otorrhœa with severe prostration. The jaundice was obviously associated with blood destruction, for pronounced anæmia not present before now made its appearance. Irregular fever, the temperature oscillating about 101° F., continued; the pulse became more frequent and very feeble. Nothing abnormal was noticed about the heart. The cough was frequent but weak. Gradually the patient grew very bad, and he died just within three weeks from his admission into the hospital. At the post-mortem it was found that the broncho-pneumonic inflammation had been so intense that it had gone on to abscess formation in several places. On the right side particularly the lung substance in places was converted into a series of smooth-walled, recent abscess cavities of various sizes. Besides this, on examining the heart recent small, red, beady vegetations were found on the valve edges. The micro-organisms found in the pus from the lung were staphylococci and streptococci; no tubercle bacilli or *Diplococci pneumonie* were discovered.

General blood infection is just as likely to happen with the pneumococcus as with other micro-organisms—indeed, it is probably commonest in the case of this organism,—but the most pernicious type of broncho-pneumonia is that due to mixed infection. It is when virulent streptococci and staphylococci occur together that the consolidation is most likely to go on to abscess formation. This breaking down of the lung substance so that pus is formed is a rare event.

5. The difficulties of diagnosing croupous pneumonia from broncho-pneumonia are augmented on account of the fact that occasionally broncho-pneumonia may be limited to one side, or even to one lobe, and, on the other hand, because sometimes broncho-pneumonia arises as a complication of croupous pneumonia. This complication is more likely to occur in a flabby or rachitic infant than in one previously healthy. In some cases the question as to the variety of pneumonia present can only be decided by examination of the lungs after death. In the following case it was not until the consolidated area had been carefully studied after death that the exact nature of the disease was determined.

The patient was a male child of eleven months. He was inclined to be puny, and there was some beading of the ribs. He was admitted into the hospital with a temperature of 103° on the third day of his illness. This had started suddenly with dyspnœa and refusal of food. On the fourth day of the disease well-marked signs of massive consolidation limited to the left lower lobe were present. A little vomiting and severe abdominal distension with undigested stools complicated the case. The fever, which had been steadily high, dropped suddenly on the tenth day. The following day another rise of temperature occurred, but lasted only temporarily; and afterwards, when all his symptoms improved and the signs began to clear up rapidly, there seemed good justification for believing the disease to be croupous pneumonia. The points in favour of this variety were the sudden onset, the early appearance of massive consolidation limited to one lobe, absence of signs of general bronchial catarrh, minor degree of respiratory embarrassment, the high continuous fever ending suddenly, and the apparently rapid resolution and convalescence. When, therefore, five days after the temperature had fallen, abnormal pulmonary signs again made their appearance, it was considered probable that they were due to the occurrence of broncho-pneumonia as a complication. The dulness at the left base, which had previously gone away, came back again; the râles became sharper and finer, and the breath-sounds, which had already regained their vesicular character, once more were of the bronchial type. At the same time, not only did catarrhal signs scattered elsewhere in the lungs make their appearance, but severe prostration also with laboured breathing, cyanosis, and recession were observed; the distension reappeared and tetany came on. Some pyrexia arose once more; the patient got rapidly worse and died.

The post-mortem appearances went entirely in favour of the view that broncho-pneumonia had been the disease throughout. Dr. Trevor, who performed the autopsy at St. George's Hospital, has kindly supplied me with the notes of his pathological examination, from which I will quote the following extract:

"*Lungs.*—The left lower lobe is solidified by

Digitized by Google

the aggregation of small areas of consolidation which surround the smaller bronchial tubes; alternating with these areas are patches of collapse. . . . Other lobes show scattered patches of broncho-pneumonic consolidation. Microscopical examination of various portions all show the same picture. The smaller tubes are the seat of a very acute inflammation, their walls infiltrated with leucocytes and their epithelium shed. In many places the lumen is filled with leucocytes and the outline of the tube lost. Around the tubes the lung alveoli are filled with leucocytes. The extent of the consolidation varies: in places only a small peribronchial focus is seen; in others, by fusion of the zones surrounding neighbouring tubes, larger consolidated tracts have resulted. But the consolidation is patchy; everywhere islands of alveoli free from consolidation—in some places merely compressed, in others showing desquamated epithelium or catarrhal cells—are met with, separating the consolidated areas. . . . No fibrin is seen in the alveolar contents."

All the above patients were treated in the wards of St. George's Hospital, and I take this opportunity of thanking the physicians of that hospital for so kindly allowing me to make use of the cases under their care.

THE action brought by Burroughs, Wellcome & Co. against certain chemists to prevent the passing off of goods not made by Burroughs, Wellcome & Co., when the trade mark "Tabloid" is used in prescriptions and orders, was concluded on December 14th, by judgment being given by Mr. Justice Byrne in favour of Burroughs, Wellcome & Co., on all counts. We are glad to emphasize the importance of this case, as it is perfectly clear that when a medical man wishes his patients to have products manufactured by Burroughs, Wellcome and Co., he puts "Tabloid" on his prescription and naturally expects that there will be no substitution of goods made by others than Burroughs, Wellcome and Co. It has now been definitely decided in the Courts of Law that a medical man not only has a moral but a legal right to demand that any prescription he issues with the word "Tabloid" in it is to be dispensed in the only proper manner. It was ingeniously suggested at the trial that Burroughs, Wellcome & Co., had sought to rob the English language by preventing its natural development in endeavouring to appropriate such a word as "Tabloid," but as the Judge pointed out it was more correct to say that Burroughs, Wellcome & Co. "have made a gift, such as it is, to the language while reserving a special use of it to themselves."

TWO LECTURES
ON
PILES, THEIR NATURE AND TREATMENT.*

By J. JACKSON CLARKE, M.B.Lond., F.R.C.S.,
Senior Surgeon to Out-patients at the North-West London Hospital; Surgeon to Out-patients at the City Orthopædic Hospital.

LECTURE II.

COMPLICATIONS OF INTERNAL PILES: *Strangulation.*—By this is meant the condition, commonly called an "attack of piles," that arises when internal piles have become protruded. Clinically large bluish tumours, surrounded by everted mucous membrane and œdematous skin, are seen, and cannot be returned by the patient. The part of such piles that is below the sphincters naturally becomes swollen from engorgement of the veins, which may rupture, causing hæmorrhage, or become thrombosed; and finally, if the condition is not relieved, sloughing of the protruded parts occurs, and thus sometimes results in a natural cure of the piles. At other times fibrous masses (white piles) are formed from organisation of the thrombosed vessels; such white piles, such as the one I now show you that I recently removed from a man of seventy-five, may cause great inconvenience by remaining prolapsed and giving rise to incontinence of wind and liquid fæces, and continuous discharge of mucus.

TREATMENT.—If the strangulation is recent the patient may be placed in the prone position, with the pelvis raised on pillows. The protrusion may be covered with wool soaked in a strong solution (20 per cent.) of eucaine, and left on for ten minutes. After this the surgeon's index finger, well anointed, should be passed into the rectum, and an attempt made very gradually to return the piles. If this fails, or if the piles are sloughing, boric acid and thymol fomentations should be applied until the sloughing is complete. If the piles have been returned the patient should be put to bed and the bowels confined for three days: if, after this interval, the general condition of the patient is good, and there is no contra-indication,

* Delivered at the Medical Graduates' College and Polyclinic, November 18th and 25th, 1903.

Digitized by Google

he may be prepared for removal of the piles; in any case a full aperient should be given on the fourth day.

Ulceration may follow sloughing of a pile. Thrombosis of a pile may occur without its ever appearing at the anus, and if ulceration follows thrombosis, as it sometimes does, a dangerous hæmorrhage may be the first sign that anything is wrong. In such cases examination of the rectum reveals an ulcer with an artery or a vein, or both, exposed at its upper part.

Complications arising after Operation.—*Hæmorrhage.*—This may happen to the most experienced of operators, but is extremely rare if care has been taken to tie the pile as tightly as possible and not to cut the stump so short that the artery may retract, and again with the local after-treatment of the wounds. If hæmorrhage occurs when no tube has been used in the dressing, and the dressing has been applied firmly, or if the sphincter has been dilated so insufficiently that it has recontracted, a patient may bleed to death without a drop of blood appearing on the surface. The hæmorrhage may be primary, recurrent, or secondary.

Treatment.—If the restlessness, pallor, clammy skin, and weak, rapid pulse, and possibly air-hungry respiration of the patient, apart from any visible escape of blood, proclaim from afar that he has bled, no time should be lost in removing the lips of the anus and allowing the blood and clot to escape. In order to assist in this the patient should be asked to make an expulsive effort. An injection of two pints or more of cold saline solution should be given; it will wash out the remainder of the blood. The part should then rapidly be examined for a bleeding point. In women the front of the rectum can be everted by passing two fingers into the vagina. If the bleeding continues and the bleeding point is not at once found, no time should be lost in giving ether; chloroform should be avoided if the loss of blood has been at all severe. The part can then be thoroughly examined, successive portions of the mucous membrane being drawn down and examined. In all probability this will reveal the seat of the bleeding, and a ligature must be securely applied. Should it fail, the rectum must be efficiently packed. If none of the special means of doing this, such as Gowlland's tube or Benton's inflatable tampon, are at hand, a "petticoat plug" can be improvised by securely tying the apex of a clean handkerchief, made cone-shaped, around a penholder, passing it four inches up the rectum, and packing plenty of cotton wool between the stem and the handkerchief. If a stiff tube can be found instead of the penholder, so much the better; it will enable the flatus to escape, and from time to time saline solutions can be injected to relieve the patient's faintness. If the latter is extreme, intra-venous injection of sterilised saline solution will be required.

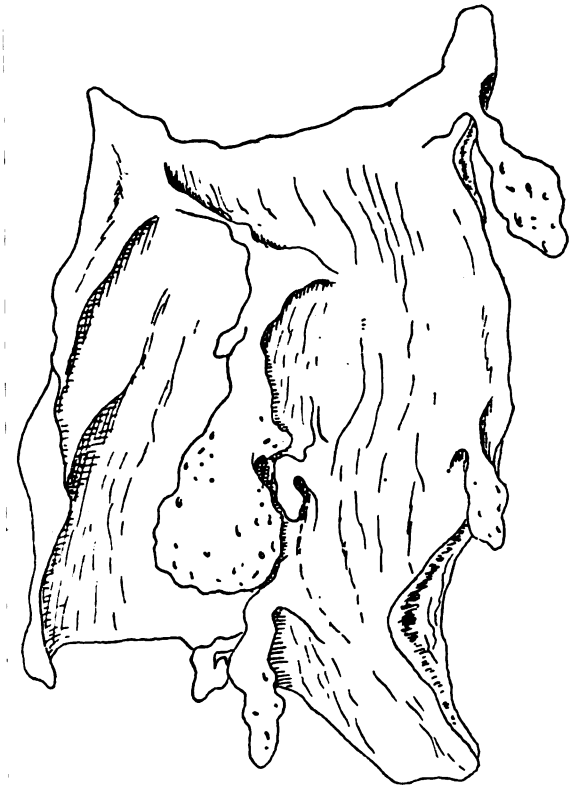


Fig. 5.—A portion of the rectum of a young woman; polypi, the result of chronic ulcerative colitis, are present.

Abscess.—Any severe pain or difficulty in defæcation felt after the initial pain of the operation is passed should be carefully investigated. An abscess will usually declare itself about the time of the separation of the ligatures (seventh or eighth day) by throbbing pain. In order to prevent the formation of a fistula this should be freely opened. Careful examination is required to ascertain the site of the abscess, whether submucous, ischio-rectal, or pelvi-rectal.

Digitized by Google

Narrowing of the anal canal.—It can hardly take place unless more than two piles have been removed. The condition will show itself by the twelfth day after operation, and, if it is found to be present by passing the index finger into the canal, can be dealt with by daily passing into the anus the whole length of the index finger, or a bougie three quarters of an inch in diameter.

Stricture.—Fibrous stricture of the rectum. I have seen this once as the result of an operation for piles, though I could not ascertain what particular operation had been done.

Pyæmia and tetanus have been recorded, but the death-rate from operations for piles is happily very small.

Complications of Piles.—If *fissure* or *ulcer* is present, the operation should be modified, complete section of the external sphincter being made instead of stretching the sphincter at the time that the piles are operated on. The pile itself may bear an ulcerated surface, which will, of course, be removed with the pile.

If a *fistula* is present with piles, both can be dealt with at the same time, the external sphincter being cut; if the fistula is a difficult one the piles can be left to be operated upon about a fortnight after the fistula.

Impaction of fæces.—Hard fæcal masses may, in spite of the preparatory purging, be found in the rectum at the time of operation. They must be carefully removed, and the rectum washed out before the operation is proceeded with. Scybalous masses may descend into the rectum when the first purge is given after operation, and then great care is required in their removal. If they are large, injections of warm olive oil should be used to soften them, and the surgeon should aid their passage with his finger.

Polypi.—Pedunculated growths, whether smooth adenomata or villous growths, are occasionally present together with piles, and if so they should be removed at the same time as the piles. The peduncle of such polypi should be carefully examined to see whether they contain any small intestine, which, when the peduncle is large and invaginates the whole thickness of the rectal wall, may find its way into the peritoneum-lined cavity.

Contra-indications to Operation on Piles.—Patients may be extremely anæmic and exhausted from continual loss of blood in cases of internal piles, and when this is the case we must be on our guard lest we conclude that the patient is too ill to bear an operation, when in reality the latter is the only measure that can restore health. On the other hand, some full-blooded persons, with a tendency to congestion of the cerebral vessels, are benefited by occasional bleedings, and the palliative treatment of their piles is the best also for their general health; so for them it is wisest to refrain from operation. During pregnancy, operations for piles are best avoided. When abdominal tumours are present the piles should not be operated upon, unless the tumour has previously been taken away and the piles have persisted.

Malignant disease.—Before any operation for piles is undertaken, careful consideration of the symptoms and examination of the rectum should be made to make sure that the patient has no cancerous growth, which, by obstructing the hæmorrhoidal veins where they pierce the wall of rectum, not uncommonly causes piles.

Ulcerative colitis.—This grave affection in its chronic form produces polypoid masses that may protrude from the anus and be mistaken for piles. Many years ago a practitioner submitted to me a portion of a rectum (Fig. 5) that had been removed after death from a young woman. The history of the case was that internal piles had been removed some months previously, but did nothing to improve the anæmic condition of the patient, who succumbed to persistent diarrhœa and gradual exhaustion after the operation. The anatomical changes in the bowel showed that the case was one of chronic ulcerative colitis. This condition begins with a general inflammatory swelling of the mucous membrane of the lower part of the colon, accompanied with ulceration. The second stage of the process is marked by most extensive ulceration, which leaves knobs and bridges of mucous membrane. In this stage it may be difficult to distinguish from a cancerous ulcer when it occurs in the rectum, and when it is situated higher up in the colon may also simulate growth, by arresting scybalous fæces in the meshes of the bridges, etc., of mucous membrane. A third stage of chronic ulcerative colitis is that already referred to, where pedunculated masses are present.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 586. WEDNESDAY, JANUARY 20, 1904. Vol. XXIII. No. 14.

CONTENTS.

PAGE

*On the Operative Treatment of the Conditions of the Large Bowel which result from Chronic Constipation. By W. ARBUTHNOT LANE, M.S. ... 209

* A Clinical Lecture on Impetigo. By T. COLCOTT FOX, M.B., F.R.C.P. ... 214

*On the Nature of the Connection between Asthma and the Nose. By ALEXANDER FRANCIS, M.B., B.C. ... 222

** Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½ *Bartholomew Close, London, E.C.*

ON THE OPERATIVE TREATMENT OF THE CONDITIONS OF THE LARGE BOWEL WHICH RESULT FROM CHRONIC CONSTIPATION.

By W. ARBUTHNOT LANE, M.S.,
Surgeon to Guy's Hospital and to the Hospital for Sick Children.

I HAVE discussed this subject briefly in papers in the LANCET* and CLINICAL JOURNAL.† I now propose to consider it in greater detail.

The cæcum and ascending colon form the cesspool of the gastro-intestinal tract, and into this portion of the large bowel the fluid contents of the small intestine are passed. In a more solid form the contents of the cesspool are passed on into the transverse colon and then through the splenic flexure into the decending colon, sigmoid, and rectum. Now the density of the material in the cæcum varies within very wide limits in different individuals and under different circumstances. In those who possess but a poor store of energy the fæces often remain sufficiently long in that portion of the bowel to become quite firm; and its density as it passes the splenic flexure, and later as it enters the sigmoid flexure, is correspondingly greater. An habitual distension of the cæcum and ascending colon, especially in people who spend most of the day on their feet, determines an inflammation of the bowel of a sufficient intensity to produce adhesions between its peritoneal coat and the peritoneum lining the abdominal wall in immediate relation to it externally. These adhesions are advantageous in that they pin down the ascending colon and cæcum and oppose its downward and inward

* "Chronic Obstruction of the Cæcum and Ascending Colon," Jan. 17th, 1903; and "An effectual means of dealing with the conditions of Chronic Obstruction of the Large Bowel resulting from the Adhesions which develop in consequence of Chronic Constipation," 'Lancet,' Jan. 2, 1904.

† "On certain Abdominal Affections," CLINICAL JOURNAL, March 25th, 1903.

Digitized by Google

displacement when loaded up with fæcal contents. Those acting upon the hepatic flexure exert such a strain when the cæcum is loaded in the erect posture as to produce a kink or a practical obstruction in this situation, offering a very serious temporary obstacle to the passage of material through the flexure.

The adhesions also involve the appendix, which is inflamed or irritated together with the cæcum. The result of this is in most cases to exert a strain upon the proximal half of the appendix, and to fix it. When the cæcum is moderately empty this attachment usually exerts no ill-effect, but any moderate distension of the cæcum produces an abrupt flexure of the appendix about its centre, and a distension of the distal segment with mucus or muco-pus. Besides, the constant interference with the free exit of secretions from this structure tends to the formation of those hard masses of epithelium resembling fæcal nodules, which of themselves may produce inflammation, ulceration, perforation, or gangrene. I am of opinion that the various abnormalities in the arrangement of the appendix are all due to the results of constipation, probably early in life during the changing position of the large bowel. Any adhesion interfering with the free movement of the appendix interferes with its function, and renders it liable to become inflamed. As you are well aware, inflammation of the appendix develops very rarely in early life, and it usually occurs only after a considerable period of overloading of the cesspool. I wish to impress on you most strongly that a daily evacuation, however satisfactory it may seem, in no wise negatives the habitual presence in the cæcum and ascending colon of an amount of fæcal contents which is excessive. By this I mean a quantity sufficient to produce irritation and the formation of adhesions which may interfere with the proper performance of the function of this segment of the large bowel, and of the appendix.

The adhesions, by connecting the ascending colon and cæcum to the abdominal wall externally, also serve to oppose the displacement of the overloaded cæcum upwards and inwards, which may occur when the horizontal posture is assumed, in consequence of which the hepatic flexure would be completely blocked. In cases of accidental over-distension in a subject who has not suffered from constipation habitually, such a displacement may occur, and a condition described as volvulus of the cæcum may result.

After the adhesions have existed for a long time there develops in their stead or in addition a mesentery, which varies greatly in its extent and in its strength. It is attached chiefly to the abdominal wall external to and above the kidney, upon which point it exerts its traction, while below it spreads itself out over the hepatic flexure and the anterior surface of the cæcum and ascending colon. The vessels running in this structure are sometimes of a size sufficient to require a ligature when it is divided.

It is only by the free division of this mesentery, and frequently of the lateral adhesions, that this portion of the bowel is freed from their constricting influence and enabled to perform its function painlessly. Even if these are thoroughly divided, unless the bowel can be made to act efficiently immediately after the operation, there naturally exists a marked tendency for the parts that have been divided to reunite, and for the obstruction, disability, pain, and tenderness to be reconstituted. Even if efficient action is obtained, for a considerable time the adhesions, etc., may be reproduced by overloading at any subsequent period.

I have already pointed out the similarity in pain and symptoms which exists between cases of stone in the right kidney and movable right kidney, and obstruction of the colon at the hepatic flexure. There is the pain and tenderness beneath the last rib, due partly to the traction exerted on this part of the mesentery in the erect posture, and by the impact of the upper limit of the distended and painful bowel in the supine position.

Again, there is occasionally pain along the course of the ilio-inguinal and ilio-hypogastric nerves, probably produced by the same causes—namely, strain or impact upon those nerves.

Also in the erect posture there is the feeling of soreness, pain, and tenderness, and of a lump in the iliac fossa which might be produced either by a congested or dilated movable kidney or by a loaded cæcum.

Owing to the accumulation of material in the transverse colon and the irritation resulting from the difficult passage of abnormally hard bulky masses through the splenic flexure, this portion of the large bowel may also become inflamed. As a consequence the mesentery which normally

Digitized by Google

supports the flexure becomes shortened and hypertrophied, dragging up the flexure and converting it, especially when the transverse colon is loaded in the erect position, into a kink which interferes more or less completely with the passage of the fæcal contents through it. The increased irritation results in the adhesion of the opposing surfaces of the dilated transverse colon and the empty descending colon, which are brought into contact by the shortening up of the mesentery and the distension of the transverse colon. This adhesion of the transverse part of the descending colon increases the difficulty experienced by the fæces in passing along, and in certain positions, especially in the erect, an actual obstruction exists.

Again, in the sigmoid flexure the overloading with hard bulky fæces produces irritation of the walls of the bowel. In consequence of this adhesions form extensively between the bowel, mesentery, and peritoneum lining the iliac fossa, so that the bowel is finally pinned down, shortened up, and rendered as straight as possible. This arrangement is very advantageous, since it enables the intestine to expel its contents more or less efficiently. If this shortening up of the mesentery did not occur, the distension of the loop would result in progressive dilatation of its lumen and in a steadily increasing elongation. This elongation and dilatation must after a time produce the rotation of the loop around the mesentery as an axis, by means of which the exit from this portion of the bowel is blocked, while the entry of material into it is not interfered with. In this manner a condition called volvulus of the sigmoid is produced, not acutely, as is generally supposed, but by the complete closure of an aperture which has been reduced progressively in size for a considerable period.

The same condition of pinning down of the sigmoid can be produced by a long-standing obstruction in the rectum, whether it be the pressure of a displaced uterus, a tumour, or a growth reducing the lumen of the rectum. I would point out that, by the careful division of the adhesions from off the outer surface of the sigmoid or its mesentery, the bowel may be raised considerably from its original level. The knowledge of this fact may be of considerable service in inguinal colotomy, or in other operative procedures on this piece of bowel.

Now, passing on to the treatment of these conditions, I, like others, operated on a large number of cases of inflammation of the appendix, and removed this structure satisfactorily as far as the life of the patient was concerned. Not uncommonly one found that the removal of the appendix had only obtained for the patient the relief that the month's recumbency, careful dieting, etc., would have produced without any operation, showing that though the appendix was affected it was not the only, or even the chief, cause of the symptoms.

While operating on a case of recurring obstruction of the large bowel at the hepatic flexure, which had been diagnosed by the patient as being due to cancer of the hepatic flexure, and by his medical advisers as renal colic, I became alive to the mode in which constipation played its part in the production of adhesions and of a mesentery which were able to interfere very effectually with the mechanics of the cesspool of the intestinal tract. It was then apparent that the condition of the appendix was entirely secondary to that of the cæcum, and to obtain complete relief from symptoms it was necessary in many cases not only to remove the appendix, but also to divide the constricting bands which controlled the lumen of the large bowel. This I did in a large number of cases, in many with the greatest advantage. I endeavoured to render this benefit as permanent as possible by insisting on great attention being paid to the bowel subsequently by the use of purges, enemata, massage, exercises, etc.

In a certain proportion of cases—and I regret to say that the proportion is not so small as I would like—a recurrence of the symptoms ensued. I then came to the conclusion that these patients could not be benefited permanently by the treatment of effects, as we had no satisfactory means of dealing with them, and that to ensure peace and comfort to the sufferers in the future the cause of all the trouble must be removed. It was apparent that these patients lost as much by the absorption of ptomaines, etc., from the fæces during their prolonged stay in the large intestine as they gained from the absorption of any nutritious and useful material. Not believing, either, that the colon plays a very important part in the present state of civilisation, I determined to divert fæcal matter altogether from this part of the bowel, to convey it directly from the ileum into the upper part of the

Digitized by Google

rectum, or into the sigmoid if the former does not lend itself readily for the purpose. I knew from the results of performing this operation for individual cases of disease, as cancer, etc., that patients of the class I allude to would not incur any unjustifiable or serious risk from the operation, nor would they be deprived of any useful function. I did not undertake the operation hurriedly in any case, but made every effort to secure the safety of the patient by simpler methods in the first instance. At first I attempted to exclude any passage of fæces into the cæcum by establishing between the end of the ileum and the rectum or sigmoid an anastomosis sufficiently large in my opinion to prevent its closing subsequently. To make an efficient anastomosis between the ileum and the rectum or sigmoid which will remain serviceable, and which will probably not permit the passage of fæcal matter into the distal segment of the former, it is necessary to build the ileum into the large bowel, so that the circumference of the combined bowel does not exceed the original circumference of the large intestine. This is done by cutting away half the circumference of the ileum, and a corresponding amount of that of the large bowel, and lacing the edges of the apertures together. I soon came to the conclusion that the only certain means of putting the large bowel entirely out of gear, when the rectum is overloaded with solid fæces, is to effect an ordinary lateral anastomosis between the ileum near its termination and the upper part of the rectum, or the sigmoid, as may be most convenient. After this is done the ileum is divided between the seat of anastomosis and the cæcum. It seems to me that this arrangement is safer than the division of the ileum and the implantation of its proximal end into the large bowel. I am sure that the principle involved in cutting off advantageously from the intestinal tract of almost the entire length of the large bowel, which to a certain class of person is a distinct detriment rather than a source of strength, will have a very extensive application in future in surgical practice. I would strongly impress its importance on my readers. Perhaps I cannot do better than illustrate the method I adopted at first by giving two cases.

M. L—, æt. 19 years, had always been delicate, and suffered all her life from constipation. When nine years old had "ulceration of the bowels." Two years ago her constipation became worse, and she suffered severely from pain and tenderness in the right loin and iliac fossa, and attacks of griping in her abdomen generally. In November, 1901, I removed her appendix, which was much inflamed and adherent to a hydrosalpinx, which was also excised, the ovary being left undisturbed. The wound healed perfectly. Very soon after her return, her symptoms returned, and she was as bad as ever. At the time I performed this operation I was unaware of the condition of mechanical disability produced by adhesions, but when she came in May, 1902, begging for the temporary relief the operation had afforded her on the last occasion, I opened her abdomen and divided as freely as possible the bands and mesentery which obstructed the ascending colon and flexure, so that I was able to lift them out of the abdomen. This operation gave her merely the same temporary freedom from pain as before. In October, 1902, I operated again, found the adhesions had re-formed, and again freed them. We administered purges immediately after the operation, and continued them. The relief obtained extended over a longer period than before, but the symptoms returned with the same severity.

In July, 1903, I operated again, put her on her left side to separate the parts to which the bands were attached, and administered purges even more freely than before, with no better result.

In October, 1903, I connected the lower end of the ileum by lateral anastomosis with the upper part of the rectum, rendering the aperture of communication very extensive, being about three inches in length. The result of this was most satisfactory. She has put on weight considerably, looks happy and comfortable, and has lost the dirty colour of her skin which was becoming a very distressing feature of the case.

The next case was that of W. M—, æt. 37 years. The history was similar to the last. In November, 1902, I removed a chronically inflamed appendix and freed the hypertrophied and dilated cæcum and ascending colon from the adhesions and bands which impaired the proper performance of its functions.

The relief obtained from the operation was only temporary, and another operation was performed, when the adhesions were again freely divided and purgation adopted. Again the condition relapsed.

Digitized by Google

On December 10th, 1903, I connected the lower portion of the ileum to the sigmoid by lateral anastomosis, making a communication of considerable size. It was not possible to employ the rectum, as it was drawn tightly down by the contraction of the omentum of the sigmoid loop, which was bound down and stretched tightly along the iliac fossa. By carefully dividing the adhesions along its outer aspect I was able to pick it up sufficiently to attach the ileum to its centre.

These two cases are sufficient to show the condition for which the operation is suggested and the mode of its procedure. At the same time, as it is conceivable that an accumulation of solid fæces in the large bowel may so occupy the aperture in its side as to render it possible for material to pass along the lumen of the ileum beyond a lateral anastomosis, however large, and so on into the cæcum, I have, as I said previously, discarded this method in favour of the complete division of the ileum, the proximal end being made to open directly, or preferably laterally, into the rectum or sigmoid while the distal end is closed. By this means the entry of any fæcal matter into the cæcum is effectually prevented. This operation presents little risk and is readily performed, and affords a freedom from almost constant pain and distress, which is often very great indeed, rendering life almost unbearable.

If there be much distension of the cæcum at the time of operation—as, for instance, when the condition described as volvulus exists,—before closing the distal aperture in the ileum the cesspool can be drained of its contents and irrigated through it, when it can then be closed.

As regards volvulus of the sigmoid, the treatment must vary to some extent with the condition of the bowel and of the patient generally. If the loop be greatly distended, its walls acutely inflamed, and the patient severely ill, a resection of the entire loop offers the best chance of success.

The following is an account of such an operation of resection of the sigmoid for volvulus, and published in the 'Medical Press and Circular,' May 4th, 1899:

"Mr. Arbuthnot Lane operated on a case of intestinal obstruction in a middle-aged woman who had been admitted under Dr. Frederick Taylor. Her abdomen was enormously distended; the bulk of the swelling seemed to be due to the presence of a large resonant mass in its interior projecting beyond the adjacent coils of intestine. The patient gave an indefinite history of attacks of previous obstruction, which had been both acute and chronic. Mr. Lane made an incision along the length of the linea alba, through which there at once leaped out a hugely distended coil of bowel; this measured 1 yard in length and 18 inches circumference; the wall of the bowel was exceedingly thick, and its mesentery opaque and scarred. Further examination showed that the condition was one of volvulus of the sigmoid. He adopted the method of excision which he had urged at a meeting of the Clinical Society of London on April 26th, 1895, and resected the whole loop, using the largest size of Murphy's button for the purpose of joining the divided ends, controlling the proximal and distinct portions of the bowel during the process by his own clamps, which are now used pretty universally. It was the largest volvulus that Dr. Taylor or Mr. Lane had ever seen. Mr. Lane said it seemed to him that this method of excision was the only scientific plan, as he had before pointed out at the Clinical Society in the discussion of the paper on 'Acute Intestinal Obstruction,' by Dr. F. L. Benham and Mr. Silcock. It was quite clear, he asserted, from the condition seen in the case he had just operated upon, that any other recognised form of treatment would not have brought about the complete restoration of function which he hoped might result in this patient. The reasons in favour of excision were, he said—(1) That recurrence is impossible. (2) That a loop which is often acutely inflamed and extensively ulcerated, and the source of immediate danger from perforation, gangrene, septic absorption, etc., is at once removed. (3) That no drainage is necessary, and no fistulous opening remains. (4) That it is a procedure to which no reasonable objection can be offered. Button was passed on 31st day, April 25th, 1899. The patient made an uninterrupted recovery."

If a case of volvulus of the sigmoid is seen at a comparatively early date, as is not uncommonly the case now, because of the much greater experience of general practitioners in abdominal conditions, it may be quite unnecessary to excise the twisted loop. The reason of this is that the sigmoid, though considerably dilated, is not suffi-

Digitized by Google

ciently inflamed but that it can readily recover if material is able to escape freely from it.

In such a condition we can but imitate the process of shortening up of the meso-sigmoid, which usually takes place to an extent sufficient to oppose the excessive elongation, dilatation of the loop, and the subsequent formation of a volvulus. To do this the loop is exposed and turned out of the abdomen and untwisted. An aperture is made into it if the material does not escape freely when it is untwisted and its contents are evacuated. The aperture in the gut is closed, and then its enormously hypertrophied outer longitudinal band of muscle-fibres is sutured to the edge of the oblique incision in the left groin, which, for this purpose, is made close to the anterior inferior spine of the ilium. Being fixed through a considerable portion of its length along its convexity, the sigmoid cannot again rotate and occlude its lumen. By adopting this method instead of excision I obtain a reasonably good working arrangement, and avoid any such risk as is entailed by the removal of the distended loop. If there is any evidence of considerable obstruction to the passage of fæces along the remainder of the large bowel, I would not hesitate to connect the ileum with the first piece of the rectum by a lateral anastomosis, and to divide the ileum beyond it.

A Contribution to Immunisation with Ovary.—Skrobansky has endeavoured to arrive at the physiological significance of the ovary and corpus luteum by immunising animals with these tissues. The reagent used was the spermatic fluid of the same race as the ovary. Rabbits and guinea-pigs were immunised with the ovaries of white mice and of rabbits, and with ovarian tissue of the cows triturated in the liquor folliculi of the graafian follicles and with corpus luteum in physiological salt solution. It was found that the sera obtained n this way all stimulated the activity of the spermatozoa of the kindred animals to a marked degree. Rabbit serum immunised with beef ovary contained agglutinins for beef spermatozoa, but after the introduction of corpus luteum these did not appear.—'Münchener medicinische Wochenschrift,' November 3rd, 1903.—*Medical Record.*

A CLINICAL LECTURE
ON
IMPETIGO.

Delivered at the Medical Graduates' College and Polyclinic,

By T. COLCOTT FOX, M.B., F.R.C.P.,

Physician for Diseases of the Skin to the Westminster Hospital; Dermatologist to the Metropolitan Asylums Board's Ringworm Schools.

GENTLEMEN,—I have selected as the subject of my lecture this afternoon the group of eruptions denominated *Impetigo*, because of the excessive frequency of these eruptions, the interest attaching to the evolution of our knowledge on the subject, and the marked advances made in recent years towards the solution of what has appeared to be a complicated problem.

When we survey a long series of skin eruptions we are struck with the frequency with which elevations of the surface containing pus are encountered. You know that these collections of pus may range in size from a pin's head to a tumour-like formation, commonly known as an abscess. They may be superficial, or more or less deep-seated. They may be obviously located about a pilo-sebaceous follicle or not. Then we note that sometimes the pus is apparently present from the start; in other lesions the pus formation does not seem to be so constant or essential, and is of later production. Thus the clear contents of lesions we know as vesicles, phlyctenæ, and bullæ may ultimately become puriform. We find it most important to draw a distinction between eruptions in which, for instance, pustules are essential elements in the eruption, and others in which the elementary lesion is a vesicle or bulla, and in which the contents gradually become puriform, as in eczema, scabies, varicella, and pemphigus; and, again, others where the pustulation is intermixed or superadded to a primary eruption, as so frequently happens in all the itching, and consequently scratched eruptions, as prurigo, lichen urticatus, etc.

In the absence of histological and etiological data, our forefathers naturally made full use of the external characters of eruptions as a basis for classification, and our illustrious fellow-countryman, Robert Willan, distinguished an order of *Pustulæ* characterised by elevation of the cuticle

Digitized by Google

with an inflamed base containing pus. This order was distinguished from the vesiculæ and the bullæ, though it was recognised that the clear contents of bullæ might become more or less puriform. The slowly suppurating lesions known as acne and sycosis were also placed in a separate order.

In this order, *Pustulæ*, five genera were distinguished, viz. *impetigo*, characterised by small yellow pustules known as psydracia, occurring in clusters and terminating in a thin scaly crust; *porrigo*, an eruption of straw-coloured pustules (known as favi and achores) concreting into yellow or brownish crusts or cellular scabs; *ecthyma*, distinguished by isolated, scattered, large pustules known as phlyzacia, each seated on a hard, elevated, red base, and terminating in a thick, hard, greenish, or dark-coloured scab; the contagious fever, *variola;* and lastly, *scabies*, which was regarded as a multiform eruption with pustules as a constituent, but the cause not yet widely recognised.

In subsequent years the varieties of impetigo were rearranged by authors, and brought by most into relation with the vesicular eruption called eczema; and the seal was set on this arrangement by the powerful influence of Hebra, of Vienna. Soon, too, the heterogeneous group of diseases included under the term porrigo was gradually broken up, and the members relegated to eczema, to ringworm, to favus, and to alopecia areata. One variety alone survived as a distinct disease with English authors, viz. the porrigo favosa of Willan and A. T. Thomson, or impetigo favosa, as Bateman preferred to call it. The contagious character of this eruption was insisted on by all. Thus Plumbe, in 1827, wrote that, "like ringworm, the porrigo favosa spreads rapidly by infection through families of children, and it is not uncommon to see several of them inoculated from one child, around whose mouth one or two pustules may have appeared, and the contents have been applied to the lips and cheeks of its brothers and sisters in kissing them. The breasts of the nurse are not unfrequently inoculated in the same manner." Startin, of the Blackfriars Hospital, in 1846 (Lectures in 'Medical Times') identifies an eruption called by him porrigo simplex with the porrigo favosa of Willan, and writes that "it is not uncommon to see whole families suffering from porrigo simplex or favosa, and I have known nearly every individual in a school of young children thus affected." It should be stated that it was not until a later period that the part played by pediculi in exciting pustular eruptions was recognised. In France the name impetigo favosa had caused confusion with the parasitic disease favus, but in 1857 Devergie recognised a contagious impetigo. By Hebra the porrigo or impetigo favosa was also gathered in the eczema net, and it was only later that Kaposi consented to recognise as somewhat special this eruption and that associated with pediculi of the head.

The Blackfriars teaching was carried on by Mr. Hutchinson, and you are doubtless familiar with the portraits in the New Sydenham Society's 'Atlas' illustrating the association of porrigo with head lice, vaccination crusts, and other states. He says by the term porrigo it is intended to imply an eruption due to local pus inoculation, and usually to be quickly cured by local remedies. The early stage, however, is usually an irregular vesication, and not a true pustule, the contents of which rapidly become purulent and dry up into thick, heaped-up, dirty scabs. It is believed to be contagious not only to different parts of the skin of the same patient, but also to other persons. It may originate from any cause which induces the formation of pus—such, for instance, as a scratch,—and the theory adopted is that this eruption is due to the transplantation of pus-cells by the patient's fingers from one part to another. When lice are present in the head, as is so frequently the case, it is probably the patient's scratching which first induces the formation of sores.* Mr. Hutchinson also pointed out that this porrigo is often superadded to some primary eruption whenever a child of delicate skin becomes the subject of any kind of pus-secreting sore. Lastly, he distinguished this porrigo from eczema by the ease with which local remedies cured it, by its contagious property, by the character of the discharge and the thick heaped-up crust as compared with the transparent crusts

* Whether traumatising of the scalp be directly due to lice or indirectly from scratching for the relief of irritation, the sites are apt to become rapidly infected with organisms, and so impetigo is a frequent complication of pediculi in the scalp. But, as F. J. Payne remarks, lice may live for months or years in the scalp without impetigo supervening.

Digitized by Google

of eczema, and by the fact that the crust covers the whole of the inflamed patch and is not surrounded by redness, which means, I take it, that the eruption does not consist of a crowd of vesico-pustules seated on an inflamed base. Dr. F. J. Payne, in the 'St. Thomas's Hospital Reports' for 1883, states that at that time in Germany Unna alone was emancipated from the Vienna teaching, and that at the Hôpital St. Louis at Paris the eruption was then recognised, but that he knew from personal experience in the year 1865 it was not so. Payne, like Hutchinson, remarks on the frequency with which this eruption originates from some suppurating wound or slight festering injury to the skin, and he calls attention to its common association with purulent inflammation of the mucous membranes or orifices of the body (conjunctivitis, otitis, vaginitis, rhinitis, or ulcerative stomatitis); and in Allbutt's 'System of Medicine' he adds that he has traced impetigo to such sources, and conversely these mucous membrane inflammations to impetigo. In the 'Westminster Hospital Reports' for 1887 I gave the common sources of the eruption as (1) pustules arising in the scalp from pediculi; (2) discharges from the nose, eyes, ears, etc., following measles, etc.; (3) discharges from festered excoriations, abrasions, and wounds; (4) scratched pustules of vaccination and varicella.

Meanwhile, in the years 1864 and 1868 Tilbury Fox called attention to an eruption which he thought had been probably included previously under that very indefinite term impetigo sparsa. He described it as a superficial eruption of the skin, and often of the conjunctivæ and nostrils, of markedly uniform character, readily yielding to treatment, and varying in severity both as regards general and local manifestations at different times. The eruption, he writes, is constituted of distinct and separate coarse vesicles (though they may be confluent on the face), which tend to enlarge in five or six days into flattened bullæ or phlyctenæ the size of a threepenny, fourpenny, or sixpenny piece, or larger if unruptured, the centre always becoming depressed and umbilicated, and the contents opalescent.

In two or three days after the appearance of each vesicle a scab commences to form, and is characteristic by reason of its flat, straw-coloured, dry, granular, and "stuck-on" appearance, and its correspondence with the area of the bulla. There is no inflammatory areola in the slighter forms. The secretion contains numerous inflammatory, and at a later stage pus-cells and homogeneous material (lymph). Beneath the scabs are little superficial ulcerations filled up by viscous secretion, and at the bottom is often a little "bouton" of lymph. Itching is only present at the outset, and is then slight. The eruption usually begins on the face, or back or top of the head, sometimes the hands, or about the seat of recent vaccination, or more rarely at the seat of cuts or scratches or other wounds. It is eminently contagious and often epidemic—the secretion furnished by the eruption being capable by inoculation of producing the disease in unaffected and healthy subjects,—and its extension is due in no little degree to auto-inoculation by scratching; therefore, by reason of the special opportunties and the absence of prevention of contagion, it is much more common amongst the lower orders and amongst children. Tilbury Fox frequently produced the disease by experimental inoculation, and he extended the inoculation to a second series. He nevertheless was impressed with the analogy of its more marked forms with such a disease as varicella, and says it is certain that the eruption appears subsequently to disturbance of the system at large by two or three days. He distinguished, therefore, between the separate influences of the inoculable quality of lesions and the systemic mode of causation when contagion cannot come into operation.

Thus both Hutchinson and Tilbury Fox recognised a discrete, and often scanty, scattered, superficial eruption, unaccompanied by notable irritation, commencing with vesicles, and distinguishable from vesicular or pustular eczema, with a special incidence in childhood, easily curable by local remedies, auto-inoculable, and chiefly so by scratching, and inoculable in other persons. Both noticed its origin in suppurating wounds. The chief variations in the descriptions to be noted are that, although he described the primary lesions to be a vesication and not a true pustule, Hutchinson laid great stress on the rapid transformation of the contents into pus and the consequent formation of thick, heaped-up, dirty scabs; and he noted the dissemination of the eruption by transplantation of pus, and its origin from any cause which induces the formation of pus. Fox, on the other hand, laid far less stress on the pus production, and

Digitized by Google

described the more purely vesicular and bullous phases with formation of correspondingly straw-coloured "stuck-on" scabs. He also brought out the tendency to excentric spread of the lesions with formation of peculiar, flat, collapsed bullæ, and he proved experimentally the inoculability of the contents of the vesicles, experiments repeated subsequently by Frank P. Foster, van Harlingen in series, and by Vidal. Fox also suspected that some cases were of systemic origin and analogous to varicella. The observations of some subsequent authors tended to confirm the latter view, but the general trend of opinion has been to negative this and in favour of the purely local origin of the eruption. There can be little doubt—at least I have always taught—that both descriptions apply to phases of the same disease, for subsequent observation has shown a wide range of variability in the aspect of the lesions and their distribution, etc. For some cases there is but little tendency to pus formation, and the contents hardly get beyond the opalescent stage. In others, again, the pus transformation may be so rapid that the impression may be readily conveyed that the lesions are pustular from the outset. The character of the scabs will vary accordingly. Then, again, in some cases there is very little tendency to excentric spread of the lesions, or even to enlarge beyond the bulk of a largish vesicle. In other cases the excentric spread is rapid and marked, so that vaccinia or the circinate lesions of herpes iris or vesicating ringworm may be simulated. This is more common in eruptions with serous contents. Or the lesions may become bulky and distended instead of collapsed, so that pemphigus bullæ are copied as portrayed in Tilbury Fox's 'Atlas.' The anatomical peculiarities of the region involved have their influence. Thus you may sometimes see very large phlyctenæ on the scalp, and I have noted a large bag of serum hanging from a finger, as if produced by a burn. Again, the multiplication and extent of dissemination of the lesions, and the rapidity of their formation, differ widely, owing either to the varying virulence of the infective agent, the facilities for contagion, or the susceptibility of the soil. It is further of importance to note that one phase may be repeated in a series of persons exposed to that contagion, whilst on the other hand one phase may give rise to a different one, and it not unusual to see different phases intermixed in the same case. The usually asymmetrical distribution of the eruption is in striking contrast to what we see in eczema. Impetigo, though most often occurring on exposed situations, can be localised anywhere, or be more or less generalised.

Tilbury Fox's papers attracted special attention to the subject in America and Europe, and a considerable literature forthwith accumulated. Impetigo, as distinct from eczema, was gradually recognised on all hands, and Tilbury Fox's impetigo contagiosa was accepted in many quarters as a distinct entity. Many authors held a distinction between this impetigo and what was called simple impetigo, and others saw in impetigo only a conglomerate of diverse affections. Duhring described a special impetigo, and Bockhart, working in Unna's laboratory, called attention to a form of impetigo intimately allied to sycosis and furuncle, and caused by staphylococci. Unna himself distinguished this staphylogenic impetigo from several other forms probably due to streptococci and other organisms. Thus in 1891 Besnier and Doyon, in the French edition of Kaposi's 'Lectures on the Pathology and Treatment of Diseases of the Skin,' defined impetigo as a term uniting a series of contagious affections due probably to a series of bacterial elements which exist in all suppurations, whatever their origin, and having certain clinical characters in common more or less pronounced.

Before passing to the next stage in the evolution of our subject, let me mention some of the additions made to our clinical knowledge in the course of years. First I will allude to various striking epidemics recorded, such as the one occurring amongst one hundred soldiers at the Beggar's Bush Barracks in Dublin in 1871, and originating amongst the children at the Curragh, and the one described by Eichstedt in 1885, where seventy-five out of seventy-nine recently vaccinated children were affected. Next I will call your attention to the record of notable cases with markedly circinate lesions, sometimes generalised. I noted two such cases in sisters in the Medical Society's 'Transactions' in 1884. And speaking of the eruption being generalised leads us to the consideration of several phases of eruptions which are now suspected to belong to impetigo contagiosa. Some time back my friend, Dr. Charles Beevor, showed

Digitized by Google

me such a generalised eruption in an adult simulating pemphigus, only the bullæ were collapsed and not tense, and showed a characteristic spreading, vesicated margin beyond the crust. The bullous phase of impetigo is said to be rather common during the hot summer months in St. Louis, U.S.A. (Engmann), and a similar eruption has been described as a complication of severe sweat eruptions in the tropics. There is a great probability that the so-called pemphigus contagiosus of some tropical regions described by Sir Patrick Manson really belongs here. Grindon ('Journal Cut. and Gen.-Urin. Diseases,' 1901) has figured generalised pemphigoid cases, and Corlett ('Cleveland Journal of Medicine,' December, 1898) records that contagious bullous impetigo exists endemically in the southern portion of the United States. He describes an epidemic in an Ohio regiment stationed in Florida in the hot season. The troops brought it back with them to Cleveland. The connection of generalised cases with ordinary impetigo seems to have been established in this epidemic. Then, again, the so-called contagious pemphigus of newly born children, which used to be common in lying-in institutions, probably belongs here also; and it is worthy of remark that in these nurslings the disease is apt to prove fatal if prompt measures are not taken to prevent large areas of skin becoming denuded. Sporadic cases of the kind are sometimes met with at hospitals.*

Lastly I will note in passing the frequent coincidence of certain special lesions in impetigo, such as the raw weeping patches behind the ears, the formation of blisters around the nails, and the form of vesico-pustule known as ecthyma. Everyone appreciates the frequency of the occurrence of ecthymatous pustules where the auto-inoculation of sero-pus is going on. I, for one, have taught for years that ecthyma is not a separate disease, but that these vesico-pustules are but a variant of impetigo. Vidal, who thought it a pustular eruption, proved that ecthyma was inoculable.

I now come to another aspect of the subject. When our methods of detecting micro-organisms improved, and the association of such organisms with pus became recognised, it was surmised that all skin purulent lesions were due to local inoculation with pus germs; and investigations began with a view to determine the infective agent of impetigo, its mode of entrance to the skin, and the causes facilitating such entrance. You will recall that the lesion from which Ogston, Passet, Rosenbach, and others first demonstrated the causal connection between pyogenic organisms and pus production was the acute abscess, cutaneous and subcutaneous, and not the chronic cold tuberculous abscess. Pus may originate, it was said, where micro-organisms are not present, and on the other hand nearly all the micro-organisms may, under some circumstances, produce pus (Councilman); and perhaps some toxins may do so also. Then it is said suppuration may result from the action of certain chemical agents, such as croton oil or iodine and bromine; and amongst micro-organisms causative of suppuration may be specially mentioned the staphylococci, streptococci, the *Diplococcus intracellularis meningitidis*, the gonococcus, the pneumococcus, the *Bacterium coli communis*, the *Bacillus pyocyaneus;* the organisms of anthrax, diphtheria, tuberculosis, enteric, glanders, plague, soft chancre; and the actinomycetes, blastomycetes, and some hyphomycetes. In determining the pathogenic mode of impetigo we must also bear in mind that suppurative lesions may arise from external inoculations—directly, as by personal contact or scratching, or mediately from any infected article of clothing or furniture, or personal use such as the razor,—or they may arise as the result of a systemic infection; and remarkable eruptions of the latter class are on record. Probably many states of lowered nutrition render the soil less resisting or more predisposed to the growth of the pyogenic organisms, and we can understand variations in the characters of the eruption being influenced by the quality of the soil, the nature, amount, and virulence of the infecting agent, its point of entrance and depth of action.

From time to time authors had carefully examined the contents of the vesicles and pustules, and the crusts, without much definite result. Amongst other organisms, hyphomycetes were detected in Germany, and I corroborated this existence of a vesico-pustular ringworm simulating impetigo contagiosa (Medical Society, 1884).

* Since delivering this lecture Schamberg has pointed out the frequency with which impetigo blisters occur secondarily in the later stages of smallpox, arising between the crusts or spreading from them. Something of the same sort is sometimes seen in varicella.

Digitized by Google

Amongst the cocci noted, Crocker in 1881 and J. F. Payne in 1883 observed chains, but the cause was generally attributed to staphylococci. Pus organisms were found by various observers in boils. In 1885 you will remember that Garré, who had been investigating the organisms found in acute abscesses, rubbed his arm with a pure culture of a third generation of staphylococci and provoked an eruption of numerous little pin-head pustules with red areolæ, seated about the pilo-sebaceous orifices; and these gradually increased to the size of lentils, and finally led by confluence to the formation of an enormous carbuncle surrounded by furuncles. He also set up a phlegmon by deep inoculation and brought about a subepidermal suppuration—the early stage of a whitlow—around his nail. Let me draw your attention particularly to the type of pustule here mentioned—the small superficial one seated about the pilo-sebaceous orifices,—as its significance will appear again later. In 1887 Bockhart also published some notable observations. He examined the pus of pustules in thirty-two cases of impetigo and always found *Staphylococcus aureus* and *albus*, pure or mixed, in the fluid or cells, isolated, or as diplococci, or grouped. He stated that clear vesicles of impetigo contagiosa were free from these organisms. He inoculated himself with a sixth, and again with a tenth, generation of *Staphylococcus aureus* and *albus*, and provoked pustules, some, let it be noted again, seated about the hairs, some about the sweat-follicles, and some not so and rather larger. There was no tendency to excentric spread in these pustules. Two furuncles formed. From the pustules the organisms were found again. He concluded that a certain type of impetigo, and sycosis, and boils were due to staphylococcus infection, and that staphylococci living as saprophytes on the skin can engender these eruptions when they penetrate the epithelial tissues, either by the follicles or by abrasions. Zuckermann, also in 1887, inoculated himself on four occasions by rubbing into his skin, scratched by his fingers, pure cultures of *Staphylococcus pyogenes aureus*, and set up pustules containing similar organisms. In 1889 Bousquet, a pupil of Professor Dubreuilh, of Bordeaux, found isolated micrococci, diplococci, and some chainlettes in stained pus of pustules. He then made a series of cultures from the pus in Esmarch and gelatine tubes and obtained staphylococci; and he inoculated his arm with a pure culture of *Staphylococcus albus* and produced pustules the size of a pin's head, but does not describe their location. These pustules were themselves inoculable, and from the pus *Staphylococcus pyogenes albus* was cultivated. Dubreuilh came to the conclusion that there was only one impetigo, and that was due to staphylococci, and that the pustules of scabies were etiologically similar.

At this date the facility with which staphylococci invade secondarily various primary lesions was recognised, and in 1892 we find Wickham argued that the staphylococci found in impetigo were not secondary invasions: he found them present in the punctate erythemato-vesicular lesions, in the pustules of all ages, in the serous fluid exuding from the red impetiginous surfaces when cleared of their scabs and pus by washing, and also in the scabs; and all of these lesions, he says, were reproduced by inoculation. He "succeeded in reproducing impetigo experimentally, not only with cultures taken from the lesions of impetigo, but also with cultures of the staphylococcus taken from different folliculites, and also from a vesicle of herpes which had become purulent from secondary infection." He advocated the use of the name staphylococcia purulenta cutanea to comprehend this group of folliculites, furunculosis, and impetigo, and probably ecthyma, whitlow, styes, suppurating hydradenites, non-trichophytic sycosis, etc., all auto-inoculable and furnishing staphylococci.

We now come to still another turn of the wheel, brought about by the recognition of the fact that, whilst the staphylococcus is easy to cultivate, the streptococcus needs special media to hold its own, and may be rapidly overwhelmed by the former It is true that various observers, such as Crocker in 1881, and Payne in 1883, and others, had noticed chainlettes of cocci; but Charles Leroux, in 1892, worked at nascent vesicles by inoculating children with the contents of an impetigo vesicle, and found micrococci, diplococci, and some rare chains present; and using liquid media (bouillon) he succeeded in cultivating the streptococcus in straight and undulating chains. The re-inoculation of such a culture produced a streptococcic impetigo. As the result of his own work and that done in conjunction with Daum, he pronounced the common impetigo of children to be due to the

Digitized by Google

streptococcus, and not the staphylococcus, which was a secondary invasion.

Then Kurth investigated an epidemic in 1893 and arrived at a similar conclusion independently. He made cultivations on agar and in bouillon. He found that the streptococcus in the early vesicle disappeared as suppuration advanced and the secondary staphylococci increased. Leroux and Kurth thought the streptococcus a special species. Brocher, of Geneva, in 1896 confirmed these results and found the streptococci fatal to inoculated mice, and he recovered the organism from their blood. He identified the organism with that of Fehleisen. Again, Balzer and Griffon, in 1897, investigated indiscriminately all the cases of impetigo presenting themselves, and found the streptococcus in all. They insist on the necessity for a rigorous technique. In using a mixture of bouillon and human serum, which conserves to the streptococcus its degree of virulence, they got a culture which determined in the rabbit a subcutaneous abscess, erysipelas of the ear, and even fatal septicæmia. Balzer and Griffon reported two cases of diphtheroid impetiginous stomatitis due to streptococci; also the subepidermic periungual early stage of whitlow so commonly complicating impetigo. Let us here note that besides erysipelas the streptococcus can set up a chronic dermatitis, according to Achalme (1892), and a form of elephantiasis (Sabouraud). It is the habitual agent of diffuse phlegmon as distinguished from the more circumscribed staphylococci phlegmon; and lastly, it is the cause of the large pustule we call the ecthymatous pustule. Thibierge and Besancon declared for the streptococcic origin of ecthyma, and this view was proved to be correct by Balzer and Griffon in 1897. We thus understand why ecthymatous pustules are so frequently seen about the body and limbs of children suffering from vesico-pustular impetigo and various scratched eruptions, such as scabies and lichen urticatus.

Kaufmann was another investigator who distinguished the streptococcic vesicular impetigo from the staphylococcic pustular impetigo. Unna's views I have already mentioned, and other observers, continuing to work by the older fallacious methods, still continued to find the staphylococcus as the cause of impetigo.

In the year 1900 appeared an elaborate memoir of great importance by Sabouraud. He emphasizes the point that staphylococci complicate other cutaneous infections with great frequency, and quickly invade, for instance, the contents of an ordinary traumatic blister; that they tend to crowd out the streptococci; and that in a primary staphylococci infection they hold the field, and allow no other organisms to make head. To prove the existence of streptococci in a microbial association without using a liquid medium of culture is, he says, difficult, and he therefore insists on the necessity in this investigation of making cultures in liquid media (ascitic fluid, or neutral bouillon, or a mixture of the two) in a pipette. He insists that the ancient clinical impetigo and its diverse varieties, which may be taken as designating all the suppurating epidermites arising by isolated points, belong to two essential irreducible types, viz. the vesicular or phlyctenular impetigo named after Tilbury Fox, due to a streptococcus, which he believes to be identical with Fehleisen's; and the perifollicular pustular impetigo named after Bockhart, due to the *Staphylococcus aureus* of Rosenbach. Each of these impetigos comprehends a certain number of varieties, of which several were formerly designated clinically under a special name because one considered them as autonomous morbid entities. The phlyctenular impetigo is a superficial auto- and hetero-inoculable epidermitis characterised by the formation of discrete clear vesicles or phlyctenæ, tending to peripheral extension, and, as a rule, soon replaced by discoid amber crusts, at the periphery of which vesication may further extend. As the contents are frequently and rapidly invaded by staphylococci they become puriform, and Bockhart's impetigo may also be interspersed as a complication. Sabouraud also allows that an inoculation with the mixed cocci can occur, and then the contents of the phlyctena are opaque from the first. We need not dwell again on other variations of this impetigo characterised by the formation of bullæ, of marked circinations, of large bags of serum, of periungual blistering, of excoriations behind the ears, and ecthyma. The streptococci in the early stages of the vesicles are only found by very careful investigation, and only become numerous in adult lesions. Then they exist in the form of rare chains of three or four cocci, more often as diplococci in the serous exudate, but in the fibrinous coagulum under the crust they occur in great abundance.

Bockhart's impetigo is, on the other hand, an

Digitized by Google

epidermitis characterised by the evolution of more resistent superficial pustules formed about the orifice of a pilo-sebaceous duct (and so centred by a hair), varying in size from miliary to the top of the little finger, without tendency to excentric spread, and containing yellow pus from the start. The lesion dries *in situ* into a crust, and from the production of some necrosis a scar is often left. Bacteriologically the pustule invariably shows globular masses of staphylococci in its upper part, and rare ones elsewhere, as well, be it noted, in the track of the follicle around the hair. A pure infection by staphylococci remains pure. This eruption is of sudden evolution and often apparently spontaneous, but according to Sabouraud it can be secondary, or provoked by various traumatic influences, such as the continued use of Vigo's plaster, the application of tar, croton oil, turpentine, thapsia, after epilation of hair, etc. Directly the skin is altered by the traumatism a microbic invasion takes place and sets up an auto-inoculable pustulation.

A frequent consequence of the presence and penetration of staphylococci in the follicular tract is that complicating folliculitis of all degrees may follow secondarily in both spontaneous and traumatic provoked perifollicular impetigo. Such lesions may range from the superficial pustule through the sycosis pustule to the furuncle and subepidermic abscess. Even a non-pustular abortive scarring folliculitis may own such a cause according to this author. Further, just as a chemical or physical traumatism can provoke an impetigo of Bockhart, so can a bacterial traumatism by creating points of least resistance, and then the staphylococcus creates its own proper lesion. Examples of this are seen in the eruption known as acne pilaris and necrotica. Like the streptococcus, the staphylococcus can also set up inflammation round the nail, and, invading the nail, be the cause of a chronic dystrophy often attributed to nervous influences.

Thus ends, gentlemen, my sketch of the evolution of our knowledge of impetigo, and of the standpoint from which we must regard this group of diseases at the present day. My lecture would, however, be incomplete did I not make a brief reference to some other important developments of Sabouraud's conclusions, which tend to throw a strong light on the mysterious problem of eczema. The confusion of many impetiginous eruptions with eczema is of long standing, and Sabouraud contends that certain figured "eczemas" with fluted petaloid crusts (petaloid eczemas), and also certain diffuse exudative dermites, are really streptococcic impetigos. He also describes a chronic dermitis which becomes partly eczematised and partly lichenised, and affirms that the special artificial dermitis, long known under the name of grocer's itch, with its impetiginised surfaces and yellow papyraceous craquelée crusts, is a strepto-dermitis. Similarly there exist forms of diffuse and chronic cutaneous infection by the golden staphylococcus; and lastly, combined forms can exist in different proportions, and this complexus passes clinically by the name chronic eczema. The phenomena described under the name eczematisation belong, he holds, to mixed, diffuse, chronic impetigo, and are caused by infection by the two microbes.

The Etiology of so-called Rheumatic Tetanus.—Racine and Bruns report a case which confirms the view that idiopathic and rheumatic tetanus do not exist. If a thorough search does not reveal the site of entry of the infection the disease may be called cryptogenetic, but not idiopathic. In this case the patient, a miner, reached the hospital, presenting the typical picture of tetanus, after five days of symptoms gradually increasing in severity. Under serum therapy he improved and was discharged cured on the nineteenth day. Not the slightest abrasion could be found about the nose or throat, or on the surface of the entire body, and there was no history of any injury. The case therefore fitted exactly the ordinary description of "idiopathic" tetanus, but on the third day of his hospital treatment the patient admitted having suffered about three weeks previously from a discharge from one ear. To relieve the irritation caused by this he attempted to clear the meatus with straws torn from the broom with which he was accustomed to sweep the floor of the mine, and which was necessarily soiled with horse dung. Cultures made from the cerumen of the ear gave positive results, and the mystery was solved. The authors, therefore, advise utmost caution before making the diagnosis of idiopathic tetanus, and recommend that aural examination be added to the routine in all such cases.—*Medical Record*, November 14th, 1903.

Digitized by Google

ON THE NATURE OF THE CONNECTION BETWEEN ASTHMA AND THE NOSE.

By ALEXANDER FRANCIS, M.B., B.C.Cantab.

FEW authorities have now any doubt that there is *some* connection between asthma and the nose, but the only point which seems to be at all generally agreed upon among physicians is that at the present time damage is being done by too free surgical interference with the nasal apparatus. When the asthmatic condition in one case is relieved by a certain nasal operation, there is a great temptation to proceed further in other cases which will not yield to similar treatment, especially as different operations are reputed by various observers to produce the desired effect. The trouble lies in not being able to tell definitely how far, or in what manner, a nasal lesion is responsible for the asthmatic state.

There are two commonly accepted theories of the connection between asthma and the nose:

1. That nasal obstruction, by interfering with the nasal respiratory function, prevents the inspired air from being either properly warmed or purified before entering the lower respiratory tract. Consequently the delicate bronchial mucous membrane is exposed to changes of temperature, and to the irritation of various inspired particles, for which it is not prepared. Bronchial catarrh results, and this in turn produces asthma.

2. That certain nasal lesions act as the sensory exciting cause of a reflex, which manifests itself as an asthmatic paroxysm.

That neither theory is satisfactory in the vast majority of cases can, I think, be demonstrated beyond any doubt. The immense advantages to be derived from proper nasal respiration, and the many ills, catarrhal and otherwise, which can be removed by rendering the nasal passages free, are responsible for much of the belief in this theory. Added to this is Bosworth's * statement that "a majority, if not all cases of asthma, have some obstructive lesion of the nasal cavity."

In the first place it is very doubtful whether the mere freeing of the nasal passages, of itself, ever relieved an asthmatic condition. Cases can be quoted in which asthma has disappeared after the nose has been rendered free by the removal of polypi, spurs, turbinated bodies, adenoids, etc., but there is strong evidence to show that it was not the restoration of nasal respiration alone that relieved the asthma.

It is the exception to find asthma as the complication of marked nasal obstruction of a permanent nature, and still more rarely is it found associated with complete occlusion of the nasal passages. Moreover cases are met with in which asthma becomes aggravated after the nasal passages are made free. As a matter of fact, those who have had much experience of asthma find that in the great majority of cases the nasal passages are mechanically free. The explanation of the belief that a majority of, if not all asthmatics, suffer from nasal obstruction may be found in the fact that during a paroxysm of asthma a patient usually has difficulty in breathing through the nose, not necessarily because of any mechanical obstruction, but because this part of the breathing apparatus is, as it were, for the time being out of gear. I know the case of a patient with marked atrophic rhinitis, who was quite unable to breathe through the nose when suffering from an attack of asthma.

In support of the second theory, viz. that nasal lesions supply the exciting irritation which starts the asthmatic reflex, more evidence can be produced, but it is by no means conclusive.

Septal spurs, crests, ulcerations, hypersensitive areas of mucous membrane, enlarged turbinated bodies, and adenoid vegetations are accredited with being asthmogenous points, and cases can be quoted where the treatment of such conditions has resulted in relief of asthma. Still it must be confessed that only in a small proportion of cases does relief of asthma follow the treatment of these conditions, and then it can only be a surmise as to *why* the treatment was effective.

The chief ground for belief in this theory is based upon the assumption that mucous polypi are frequently found associated with asthma, and that in the majority of such cases the asthma can be relieved by the removal of the polypi.

If, however, a large number of cases be examined, it will be found that the association is neither as close, nor are the results obtained by the removal

* 'Diseases of the Nose and Throat,' New York.

Digitized by Google

of the polypi as satisfactory, as is generally supposed. Thus Schmiegelow * found that among 139 patients with mucous polypi only 31 had asthma, *i. e.* about 23 per cent.; while Böcker † found only 9 suffering from asthma among 310 patients with nasal polypi—less than 3 per cent. Conversely, the records of 543 cases of asthma which I possess show that 43 had nasal polypi, *i. e.* less than 8 per cent.

Although it is stated that cases in which mucous polypi are found are the most hopeful of all for intra-nasal treatment, still, as far as we can tell from any published records, the proportion of satisfactory results is exceedingly small. That polypi do *not* commonly supply the exciting cause of the asthmatic reflex is to be inferred from the following acknowledged facts:

1. That the removal of polypi by no means always relieves asthma.

2. That not infrequently the asthmatic condition becomes aggravated after polypi are removed.

3. That occasionally asthma makes its first appearance after the removal of polypi.

4. That a marked insensibility of the nasal mucous membrane is nearly always found associated with the presence of mucous polypi.

In addition to these facts my experience is that in polypi cases the best results as regards the relief of asthma have been obtained by cauterising the septal mucous membrane without removing the polypi, and that the prognosis as regards the permanent relief of asthma is less hopeful in polypi cases than in any other class.

It is reasonable to expect that the highly sensitive nasal mucous membrane may at times supply the site of the sensory irritation which starts the asthmatic reflex, and certain apparently unmistakable instances could be quoted which show this to be the case. If, however, this comprises the only connection between asthma and the nose, the ordinary asthmatic has little to hope for from intra-nasal treatment.

Far too many cases are on record where asthma has been relieved by intra-nasal treatment for all to be merely coincidences, and I believe that it will be found that there is a connection between the respiratory centre and the nose which is more intimate and important than can readily be appreciated.

We have long been led to believe that there are various kinds of dyspnœa. We are accustomed not only to distinguish between asthma arising from different causes (*e. g.* spasmodic, gastric, bronchial), but also to maintain that there are different kinds of breathlessness which must not be confounded (*e. g.* cardiac dyspnœa and spasmodic asthma). But undoubtedly it is a morbid activity of the respiratory centre which is really responsible for all dyspnœa (except in cases where disease of the lungs or other organs supplies a direct mechanical impediment to respiration), and therefore all asthma is fundamentally the same, however different it may appear according to the nature either of its exciting cause or of some prominent complication. It cannot be contended that a glass of beer, the presence of a cat,* or morbus cordis is in itself sufficient to cause asthma without the other necessary condition of an unstable respiratory centre. Yet we are asked to believe that gastric, spasmodic, and cardiac asthma have nothing in common, whereas they are fundamentally the same, and differ only in the nature of the exciting cause. If, then, the connection between asthma and the nose be, as I believe, one between the respiratory centre and the nose, and further, if, as there seems good reason to believe, the stability of the respiratory centre can be more or less controlled by treatment of the septal mucous membrane, far-reaching effects should be achieved.

I have been led to this belief by the results of treatment in a large number of cases, extending over a considerable length of time.

A little over seven years ago I cauterised the septal mucous membrane of a patient who had suffered from asthma in a severe form for between three and four years. His asthma was relieved at once, and up to the time when I last heard from him, at the beginning of this year, there had been no return. At the time I considered the cessation of asthma merely a coincidence, but obtaining some equally good results in other cases, I began to experiment freely. Among the more interesting

* 'Asthma considered specially in Relation to Nasal Disease,' London.

† 'Deutsch. med. Woch.,' 1886, No. 26.

* Certain asthmatic subjects have an attack of asthma immediately on coming into the proximity of a cat, although they may be otherwise quite unaware of the presence of the animal.

Digitized by Google

cases were some from whom I had previously removed large septal spurs without relieving asthma, although the nasal passages had thereby been rendered free. On cauterising the septum relief of asthma was now obtained in every case, and in all such cases whose histories I have been able to follow there has been no return of the trouble.

I had opportunities of trying the effect of this treatment in cases where the causes of the asthma were apparently gastric, bronchial, cardiac, or were dependent upon some obvious irritation. In each class I got instances of the complete arrest of all asthmatic symptoms. By a fortunate circumstance, in the earlier cases the relief of asthma was obtained very speedily, so that I was encouraged to persevere—for months when necessary—in other cases which showed no sign of yielding, and in very many instances complete relief was ultimately obtained.

The records show that among 543 cases of all kinds of asthma treated by cauterising the septal mucous membrane (in addition to such other accessory treatment as was obviously necessary) 316 obtained complete relief, 157 great relief, 15 temporary relief, 16 slight relief, 24 no relief, and 15 were lost sight of without anything definite being known as to the result.

With regard to the permanency of the results in the majority of the cases it has been possible to follow the subsequent histories, and in many instances the relief has been tested for more than five years. From the study of these cases many interesting facts have been elicited, but none are more striking than that the least satisfactory results as regards permanency have been obtained in the cases which were associated with mucous polypi, and the best results in cases where the nose was apparently normal. The fact that the results have been almost uniformly good in cases where no nasal lesion existed, whatever has been the apparent exciting cause of the asthma, points strongly to the inference that it is the stability of the respiratory centre which is affected by the treatment. For the only thing common to all the cases was an unstable condition of the respiratory centre. It is a noteworthy fact that in many cases the asthma has been relieved in spite of the impossibility of removing the obvious exciting irritation.

There is evidence that this connection between the respiratory centre and the nose exists in the normal state. Thus in any nose, painting the upper portion of the septum with a solution of cocaine usually produces a subjective feeling of freedom in the passage on that side, out of all proportion to the actual increase in space resulting from the action of the cocaine. This is more noticeable in cases of partial nasal obstruction, when frequently it will be found that greater freedom in breathing is experienced from painting the septum with cocaine, thereby making no appreciable difference in the mechanical freedom of the nasal passages, than from reducing engorged turbinated bodies by the application of adrenalin.

In the same way a striking effect can usually be produced in a patient suffering from chronic dyspnœa who has difficulty in breathing through the nose. As a rule, painting the turbinated bodies with adrenalin will give no relief, but if instead cocaine solution is applied to the septum, the patient will express great satisfaction at being able not only to take a deep breath, but also to breathe freely through the nose.

The facts that as a rule an asthmatic subject has difficulty in breathing through a mechanically free nose when suffering from an attack of dyspnœa, and that the disability can be removed by the application of cocaine to the septum, point to the conclusion that a morbid respiratory centre has an inhibitory action upon the upper respiratory tract, and offer the suggestion that possibly it may act in like manner upon the lower respiratory tract; and therefore it is possible that none of the commonly accepted theories may be the correct explanation of the cause of dyspnœa in the asthmatic paroxysm.

We have received from Messrs. Allen and Hanbury samples of the "Allenburys" Milk Food Chocolate. It contains 25 per cent. of the "Allenburys" Milk Food No. 1, and is intended to be used as a concentrated and digestible form of nutriment, and incidentally as a wholesome and delicious sweetmeat. This sweet is excellent as regards nutritive quality, flavour, and finish, and members of the profession will have the opportunity of recommending a sound and genuine article for consumption by children and women.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 587. WEDNESDAY, JANUARY 27, 1904. Vol. XXIII. No. 15.

CONTENTS.

PAGE

*A Clinical Lecture on Spleno-Medullary Leukæmia. By F. W. MOTT, M.D., F.R.C.P., F.R.S. 225

*The Diagnosis of the Cause of Acute Abdominal Pain. By CHARLES A. MORTON, F.R.C.S. 232

*A Demonstration of Cases at the Medical Graduates' College and Polyclinic. By BYROM BRAMWELL, M.D., F.R.C.P.E.... 237

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½, *Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

ON

SPLENO-MEDULLARY LEUKÆMIA.

Delivered at Charing Cross Hospital.

By F. W. MOTT, M.D., F.R.C.P., F.R.S.

GENTLEMEN, — This patient was admitted to Charing Cross Hospital on the 10th of this month, complaining of an enlarged abdomen. He noticed that his abdomen had increased in size so that he could not fasten the top button of his trousers, and that excited in him a suspicion that there might be some disease about him. He noticed that the swelling was hard; therefore he could not believe that it was due to any increase of fat; in fact, he said he was wasting. And that is the usual history of these cases; they waste, although the belly is getting bigger. Many patients never know that they have this swelling. I remember one case which was in the hospital who came because he had difficulty of breathing, and signs of pulmonary congestion were found, for which he was sent in. It was when I examined the abdomen, and still later examined the blood, that I detected the true cause of the disease. Now, of course, the difficulty of breathing may arise from two causes: first of all, there is the mechanical effect upon the lungs of this large splenic mass in the abdomen, pushing the lungs up and interfering with the action of the diaphragm; and secondly, there is the altered condition of the blood. Another case I remember seeing came because he had a large swelling under the scapula. It was a very unusual thing, but it was a hæmorrhage occurring in a man suffering with this disease. Another I remember came with hæmatemesis.

With regard to the bleeding, that is one of the early signs; epistaxis is an extremely common occurrence in this affection. But a patient would probably not come to the doctor simply because

Digitized by Google

of his nose bleeding, unless it was very profuse or occurred very frequently.

I shall now read you the notes of another case which was in this hospital about three years ago, because it was a very unusual case. It was a case of spleno-medullary leukæmia. He did not come to the hospital because of the tumour in the abdomen, because he had no knowledge of it. He had probably had this tumour for some considerable time. He said that he had frequently had attacks of influenza. Now influenza, as you know, covers a multitude of things very often; and if doubtful, and a man is suffering from fever and malaise, and nothing else is found, influenza is often diagnosed; and, of course, it is an extremely common complaint. I have no doubt that the later attacks of influenza of which he complained were really symptoms of the insidious disease which he suffered from. But he really came to the hospital for nervous symptoms; he came because he staggered in his gait. He walked somewhat in the same way as a drunken man, and he had a tendency to fall towards the left. There was, in addition, deafness in the left ear. I will read the notes of this case because it is so very instructive. It is instructive, in the first place, because the patient was a male, and our present patient is a male, too; and males suffer from the disease twice as frequently as females. Again, the period of life at which the disease commenced was about the same in each; this man is thirty-five, and the man whose case I shall read was thirty-eight when he died,—that is to say, the middle period of life, although cases may occur at any period of life between infancy and old age. Cases of this condition have been recorded in young infants and in old people, even up to seventy years.

With these few introductory remarks I will read the notes of this case. E. P—, æt. 38, a married man, and a wood-carver by occupation, was admitted under my care into Charing Cross Hospital on November 10th, 1899. He sought admission for enlargement of the abdomen, inability to walk, weakness of the legs, and pain in the occipital region. The family history was as follows:—The patient's father died at the age of 51 from rheumatic gout and dropsy. His mother was alive, æt. 79. She was a strong, healthy woman up to the age of 70, but since then she had been confined to her bed owing to loss of power in the lower extremities. He had a sister æt. 47, in good health, married, with a family of seven or eight healthy children. The patient had been married fourteen years; his wife was 38 years of age; she had had no children and no miscarriages. Past personal history:—The patient had always been very temperate in his habits, and had often abstained from alcohol for long intervals. He never had a serious illness until 1889, when he had an attack of influenza, and this had been followed by many similar attacks. The last occurred two years ago, was very severe, and permanently affected his health; and whereas he had previously been a good walker, he had since then been unable to enjoy this form of exercise. He utterly denied venereal disease or the possibility of infection, and he had never had malaria.

Those questions were put for a very definite reason, and when you come to consider the previous history and the family history of the two cases, you will be able to point the moral—that we are in the dark as to the causation of leukæmia.

The history of the patient before you is as follows:—

> Family history:—Father alive and well, brothers and sisters living and healthy; all relations in good health. Several children living. The patient was an officer's servant in the Royal Navy, and nineteen years ago he went to India, where he was stationed about four years. After leaving India he had fourteen voyages to Australasia, and he always kept well. The patient (according to the notes) has never suffered from syphilis or malaria.

His having been abroad of course made one think of both those diseases. Gowers, in his article in Reynolds' 'System of Medicine,' collected a large number of cases of this disease, and he found that 30 per cent. have a previous history of malaria. So malaria does seem to have some causal relation to the disease. We know that malaria causes enlargement of the spleen. You get the ague cake, and therefore it is very possible that this may be one of the causes of the disease. I find the notes are not, however, quite reliable, for had the body been more carefully examined typical pigmented specific scars would have been discovered on both shins. Syphilis in this case may therefore have played an important

Digitized by Google

etiological *rôle*. I say "may" rather than has played an important *rôle*, for with a disease so common as syphilis coincidence may be confounded with cause.

The patient's present work is that of hall-porter, which is very light and easy. He says he was never a heavy drinker or smoker, and has not smoked for the last three months, but has been taking a small amount of whisky and water daily. About three months ago he noticed his abdomen enlarging. Accordingly he went to a doctor, who gave him some medicine, after which he felt much better, and the abdomen ceased increasing in size. But when he left off taking the medicine the abdomen enlarged again. That is very probable. The patient says he has never suffered pain, but has been losing weight, and during the last week he has had sweats. He has had a good appetite, and has had slight epistaxis on several occasions.

You see the family history and previous personal history of one of these patients indicates very little as to the causation of the disease; in fact, we are really in the dark as to the cause of leukæmia. Some cases have been attributed to malaria, as I have said, and some cases have been regarded as being due to syphilis. The disease has been noticed to occur at the climacteric period in women, and one or two cases have been recorded where it has occurred during pregnancy. But, considering the whole facts of the case, we may say we know very little about its etiology or the causation.

I will now return to the notes of the first patient, E. P—.

The onset of the illness was as follows:—For the past two years the patient's health has been very indifferent; six months ago he was rather suddenly seized with violent pain in the front and left side of the abdomen, from which he recovered under medical treatment.

Now that is not an uncommon thing. These patients get a sudden enlargement—it may be a hæmorrhage into the spleen, or it may be an inflammatory condition of the spleen, for you sometimes find adhesions to the diaphragm or to the abdominal wall, and there is friction from that, and other signs showing inflammation. A few weeks after his recovery he was suddenly seized with pain and numbness of the back of the head. All these sudden conditions which come on are due probably to the altered condition of the blood, producing hæmorrhages or blocking of vessels. When we come to speak of the pathology of the disease you will better understand why these conditions should come on.

The pain was accompanied by deafness in the left ear and simultaneous loss of control over the movements in the lower limbs, with giddiness and inability to stand. During the month previous to his admission into the hospital his condition gradually became worse. On November 10th, 1899, when he was admitted, the following notes of his condition were made:—Patient is confined to his bed. His face presents an anxious, careworn appearance, and is slightly cyanosed. He complains of a dull heavy feeling over the region of the liver. Temperature, 97°; weight, 9st. 10 lbs. The abdomen is distended, slightly more on the right side than on the left; there are no enlarged veins. Girth, 87·5 cm. The abdomen is largely occupied by a firm mass in the position of the liver. It extends down to a line drawn from the tip of the ninth left costal cartilage, across the abdomen below the umbilicus, towards the right anterior superior spine. The surface of this swelling is smooth, and dull on percussion. The liver dulness extends upwards as high as the fourth interspace in the right nipple line, and to the left is continued into the præcordial area. In the left hypochondriac and left lumbar regions there is another firm smooth mass, extending forwards as far as the left nipple line and downwards to just below the level of the umbilicus. This can be pushed forwards from the loin as one solid continuous body, and is dull on percussion. The splenic notch, however, could not be felt. (So he had enlargement of the liver and spleen, and that is not infrequently the case.) Cardio-vascular system:—The cardiac impulse is situated in the fifth interspace in the nipple line. Heart-sounds are normal. Pulse, 92 per minute, moderate tension. Radial artery a little thickened. Respiratory system normal. The urine is acid, normal in colour, no obvious naked-eye deposit, and containing a trace of albumen.

Very frequently in these cases you will find a trace of albumen, and there is a trace in the case before you. Frequently you will find an excess of uric acid, and this is no doubt due to the nuclein of the corpuscles undergoing dissolution and producing uric acid.

Lymphatic system:—The inguinal, auxiliary, and cervical glands are enlarged, firm, quite discrete, free from tenderness, and freely movable. The superjacent skin is normal.

The enlargement of those glands is important, because, you see, there was a lymphatic condition

Digitized by Google

as well; and that leads me to say that there are two varieties of this disease affecting the blood-forming tissues of the body. In some cases you have the lymphatic glands enlarged from the beginning, and forming the most important feature of the disease. That is called lymphatic leukæmia. And then you have a different condition of the blood. These cases are of the spleno-medullary form, in which the bone-marrow is particularly affected. In others the spleen is largely affected and the bone-marrow not so much affected. But the commonest form is for the bone-marrow and the spleen to be simultaneously involved, giving rise to spleno-medullary leukæmia. Examination of the blood will tell you the usual form of the disease, and also which is the predominant feature, as you will understand when I come to speak of the blood.

It is particularly to the nervous system in the case admitted in 1899, and of which the pathological changes post mortem were studied, that I wish to draw your attention.

Muscular power unimpaired, as he is able to flex the knee against considerable pressure with my hand. No wasting of muscles. Co-ordination in movements of upper extremity normal, also of the lower extremities, while lying in bed. There is no hypotonus of hamstring muscles. Patient walks with a wide base, and has great difficulty in maintaining his balance. He walked like a case of cerebellar disease, oscillating to and fro like a drunken man. When I asked him to stand up and shut his eyes he reeled towards the right, and would have fallen if he had not been caught. The sensations of touch, pain, and temperature are normal; superficial reflexes normal; knee-jerks absent on both sides; micturition and defæcation normal. The ocular symptoms were as follow:—Pupils equal, 7 mm. in diameter when adjusted for distance. React to accommodation. Light reflex sluggish, and often followed by some slight secondary dilatation. There is no strabismus and no ptosis. Sight has been good until fourteen days ago, since when it has deteriorated. At the present time he cannot read because the print seems blurred. He can only dimly see a clock on the opposite side of the ward, but cannot tell the time by it.

These patients frequently have retinal symptoms; the reason is that the delicate blood-vessels of the retina get blocked up by the altered corpuscles of the blood.

The colour-vision is normal. Ophthalmoscopic examination was made by Mr. Griffiths. Right eye:—Outline of disc invisible. Vessels distended and tortuous, not interrupted, paler than the rest of the fundus. No definite hæmorrhages visible, though probably some are present. Left eye:—A white patch of indefinite nature can be seen. Hearing was affected at the commencement of his illness, but soon improved, so that the patient did not complain of deafness in either ear. However, during the last three or four weeks the patient has suffered from deafness in the right ear. A watch is heard by the left ear up to a distance of 10 cm., but cannot be heard at all by the right ear. A tuning-fork applied to the forehead in the middle line is heard best in the left ear, whether the finger be applied to the external auditory meatus or not. Taste and smell are unaffected. The mental condition is unimpaired, except during the drowsy, stuporous attacks from which he frequently suffers.

With regard to the blood examination, you will notice that the description of the blood examination in this case corresponds very closely with the blood examination of the case which we are considering to-day. In examining the blood you should always make three examinations: one for the variety of the corpuscles which exist in it, that is, a film preparation stained by suitable reagents, which will disclose the different histological elements which may be found. Secondly, you should count the corpuscles, red and white, by the hæmacytometer; and you should estimate the amount of hæmoglobin or colouring matter in the blood by the hæmoglobinometer. That was done in the case which I have been reading to you, and it was done in the present case. In the case of E. P— the red corpuscles were 1,530,000 and the white corpuscles 1,150,000, so you see the red corpuscles had diminished quite one third, and the white corpuscles had increased to almost the same number as the red. Usually, you know, in normal blood, there is one white corpuscle to 400 or 500 red, and the white corpuscles which one finds are mostly polymorphonuclear. The hæmoglobin in this case was 54 per cent., so that the red corpuscles had rather more than their value of hæmoglobin. I should doubt the accuracy of this estimation, because the hæmoglobin value is generally reduced more than that. The blood films showed a great increase in the number of white cells, chiefly leucocytes with multipartite nuclei; very few lymphocytes; some eosinophile

Digitized by Google

cells; also a great number of large pale uninucleated cells, the myelocytes of Ehrlich. When the film is stained with methylene blue and eosin the nucleus is coloured a faint blue and the rest of the cell a faint bluish pink, showing a neutrophile reaction. That is their characteristic, that the body of the cell has a neutrophile reaction. There were also some nucleated red blood-corpuscles.

The patient's condition practically remained unchanged for some time, but during one night he must have had another hæmorrhage into the internal ear, for there was deafness in his left ear; he could only hear the same watch when it was in contact with the left pinna, and he could only hear when spoken to very loudly. Practically he became deaf on both sides, and semi-comatose. The condition changed a little; next day he became a little brighter, but subsequently became much worse. He became unconscious, eventually comatose, and died. About the rest of the case I will speak to you presently.

Now we will turn to this case before us. I would like you to come and examine the abdomen of this man. I have already told you the history of the case—that it is not a very definite history. He did not complain of much beyond the enlargement of the abdomen, which he thought must be wrong, and that led him to go to the doctor; but he does not feel at all ill, neither does he look ill. His appearance is very good; he does not even look anæmic. Some of these patients are very anæmic. He is able to walk about, and enjoys life pretty well. The physical signs in the chest reveal very little abnormal. The heart and lungs are practically normal. There is no difficulty of respiration, there are no signs of compression of the lung, nor has he had any evidence of serious effects of hæmorrhage. He has had no diarrhœa, vomiting, or hæmatemesis, and the only epistaxis he has had has been slight.

He does not complain of any visual defects, although Mr. Treacher Collins finds the following condition:—The fundus: right and left choroidal reflex somewhat pale; retinal vessels, both arteries and veins, very tortuous; veins dilated, arteries pale, with bright central reflex; retinal hæmorrhages both at macula and at periphery. So you see he has hæmorrhages in the retina. There is an oval, dense hæmorrhage at the right macula, with small, light-coloured spots round it; some bright spots in left macular region. So you observe it is important in this disease to look at the retina, and you will see there what happens in other parts of the body, and may happen in the brain or in the nervous system, namely, hæmorrhages. But with all these hæmorrhages he does not complain of any visual defect, so you cannot judge of the condition of the back of the eye simply by the subjective condition of the patient. It shows how very necessary it is always to make an ophthalmoscopic examination.

With regard to the abdomen, there is an *obvious* fulness on the left side; there is *really* a fulness on both sides, but more marked on the left side than on the right. Both flanks bulge. The umbilicus is slightly protruded, but there is nothing suggesting a dropsical condition. The abdominal wall moves freely with respiration; there is no marked increased frequency of respiration. On palpation a solid mass can be felt on the left side, extending well back into the flank and projecting under the ribs, obliquely across to the right of the umbilicus, and extending down almost to the pubes. It has a distinct edge, which can be felt all round, and if you lift it you can move it forward with the hand from the flank. The notch can be felt in the upper part. It moves on respiration. It is absolutely smooth over the surface and dull on percussion. The liver also is somewhat enlarged. The liver dulness only extends above to the lower border of the fifth rib, but it extends about three fingers' breadth below the margin of the ribs. The splenic tumour is quite painless. And that is usually the history. Sometimes, however, the patient says it is painful. The apex of the heart is not displaced, but is in its normal situation. The pulse is rather full, soft, compressible, of normal rhythm and rate.

While you are examining it I will describe to you the condition of the blood in this case, and you will be able to examine a specimen of it under the microscope. In every case of this sort, when you get enlargement of the spleen, and, in fact, in all cases, you can learn so much from the blood; you should always make a microscopical examination of it. So many diseases of the blood can be revealed by this means that the microscope should always be employed. This disease is absolutely certainly diagnosed by the condition of the blood. One finds in the blood a great increase in white corpuscles, and that you would be able to discover

Digitized by Google

if you only pricked the finger and examined the blood from it in an ordinary manner. Immediately you had done that you would say this requires further investigation, even though the patient is not anæmic. You would examine and see how many red and how many white corpuscles there were by the hæmacytometer, and also take the quantity of hæmoglobin by the hæmoglobinometer. Finally, you would take film preparations and see the elements which are present in the blood. You would then find a large number of constituents which are abnormal, viz. large white blood-corpuscles with a large nucleus, and the protoplasm around containing neutrophile granules; for the disease is, as I said before, one in which the marrow of the bone is affected as well as the spleen. You find these large cells in great abundance, because they are formed in the marrow of the bone. Secondly, you find an increased number of polymorphonuclear leucocytes, that is to say, leucocytes with a twisted nucleus or multipartite nucleus. Then you see also transitional cells between these and the marrow cells. And besides, you frequently find, especially if the lymphatic glands are enlarged, and in proportion to the abundance and enlargement of the lymphatic glands, an increase of the cells which are formed in them, namely, the small mononuclear leucocytes. In fact, in lymphadeno-leukæmia you have an increase of white corpuscles depending entirely upon these mononuclear leucocytes, the other forms of white corpuscles not being increased. Besides that you will find the red corpuscles greatly diminished in numbers, and owing to the destruction of the red corpuscles and the changes in the marrow of the bones, a large number of nucleated red corpuscles may appear in the blood. The appearance of the nucleated red blood-corpuscles is very unfavourable. They are called normoblasts.

With regard to the diagnosis of this disease, it cannot possibly be mistaken for any other condition if you find an enlarged spleen combined with that condition of blood which I have described, that is to say, if you take the trouble to look. There are more mistakes made from not looking than from not knowing. Of course, in general practice a man has not the same opportunities that we have for examining patients, and very often a physical examination is not made. The doctor in this case did diagnose the disease, as he examined the abdomen and found what was the matter. But if the examination be not made, we find that the patient has been suffering from a disease for a long time, and it has been undetected. If you find enlargement of the spleen, sometimes accompanied by enlargement of the liver or of the lymphatic glands, and this condition of blood, you may be sure of what you have to deal with. With regard to the bones, I have not yet said anything about them. The case which was in here before, and whose case I read to you, had marked changes of all the bones in the body, but he did not complain of any tenderness, nor was there any external evidence of disease of bones. But sometimes you may have actual swellings of the bones, which may be very tender to the touch. Sometimes there is tenderness over the sternum, sometimes over the long bones, due to changes in the bone-marrow. But we can find nothing of that sort in this case. That, however, does not preclude the possibility of the bones being profoundly affected, as the examination of the blood revealing this large number of myelocytes indicates.

I will speak of prognosis presently.

With regard to treatment, I have to say that the best drug of all undoubtedly is arsenic, in pretty large doses. Very often the spleen will diminish in size under the administration of arsenic. You should also feed the patient up and put him under the best possible conditions for his general health. It is also of some importance to keep him from worrying, so that he may properly assimilate and digest his food. Nothing interferes so much with the metabolism of the body as worry, and therefore there is nothing which so much interferes with the nourishment of the body. So that, although he has got a serious disease, we must try and cheer him up as much as possible. These patients do frequently get very much better, and the outlook is not so hopeless as many people have said. *This* man's condition is a very hopeful one indeed. Iron is given with success in some cases, but I think arsenic is the most valuable. We shall commence with ♏v of Liquor Arsenicalis three times a day after food, well diluted, and watch the symptoms carefully. We shall have a chart made of the splenic enlargement, bearing in mind that sometimes the spleen varies in size during the course of the day. It is necessary in making observations

Digitized by Google

of this kind always to do it at the same hour each day, because before and after meals the spleen changes, and you may attribute a diminution to treatment which is really due to physiological action.

Another thing I would call your attention to is the fact that one has to be particular about the bowels, and keep the stomach free from congestion. Congestion of the stomach, and also of the mucous membrane of the bowel, is apt to occur, partly because of the mechanical conditions, and partly because of the disturbances of circulation; also to the difficulty with which this altered blood flows through the small vessels. You must be careful as to what sort of purgatives you give, therefore, or you may set up profuse diarrhœa. You should take care not to give any indigestible food, because congestion of the stomach can set up a condition which might lead to hæmorrhage and hæmatemesis. In giving arsenic in these blood diseases, you know there is the possibility of causing symptoms of arsenical poisoning. These are sneezing, vomiting, alterations in the skin and mucous membranes, itching of the eyes, running of the nose, and so on. I remember a case of pernicious anæmia which was in this ward, to which we gave large doses of arsenic. All the time that was being given the man did splendidly, but we had to abandon it because it produced in him symptoms of arsenical poisoning. He had vomiting and a silvery condition of his tongue, and definite signs of gastric irritation, which prevented us going on with the drug. As soon as we stopped the arsenic the blood-corpuscles rapidly diminished, and eventually the case proved fatal. But, as in pernicious anæmia, very often these cases, without any drugs whatever, seem to improve for a time; and, of course, bearing that in mind, we may sometimes attribute to the drug what is really due to Nature herself.

This man has not had pyrexia (except on one occasion 100°), but it is not uncommon for these patients to have remittent attacks of pyrexia in which the fever varies from 100° to 103°, lasting a few days. That condition should always put you on the *qui vive* for the possibility of this disease being present, or pernicious anæmia, or some other progressive blood affection. I will now have the patient removed.

I thought it very desirable not to speak of the prognosis in the patient's presence. You can often do far more harm than all your drugs will do good by stating before the patient the serious nature of his disease. If it is true, as I said, that worry is a serious thing for these patients, because of its interference with proper assimilation and the metabolism of the body, it would be absolutely wrong to discuss before him the serious nature of his complaint. He probably will not survive very long, although apparently he is now in a fairly good condition. Most of these cases are fatal within a few years. Now, if I had said that before him, what would have been the effect? He would have worried for the rest of the day probably, and might not have slept to-night, and in consequence we might have found him very much worse in the morning. Some cases, however, do make a recovery, and recover so far that they go about their occupations again for a time. But then a relapse occurs. As I said just now, drugs sometimes favourably influence the course of the disease. We can estimate how the disease is progressing by a periodical examination of the blood, and by examinations of the abdomen, and the size of the spleen. A diminution in the size of that organ, or an increase in the number of red blood-corpuscles with a diminution of the white, would indicate a favourable change.

Let us now devote a short time to the morbid anatomy of the disease. The disease was first described by Virchow and Hughes Bennett, almost simultaneously. And Hughes Bennett gave it the name leucocythæmia, because he found the blood filled with these white cells, which he thought were pus-cells. They do not look unlike pus-cells. In fact, when the heart was opened by Virchow in the first case he saw, there was a lot of soft, purulent-looking clot in the auricles. There was such a large quantity of this that they thought it was abscess. It was really due to this altered condition of blood. The spleen is enlarged in many cases to an enormous degree. It has been found to be from five to fifteen pounds in weight, and even more. The liver also is sometimes markedly enlarged. The marrow of the bones is changed, the compact tissue being absorbed. And not only is the marrow increased, but it is altered in appearance. It may be of a dirty rusty red colour, or of a dirty yellowish colour, like the core of an abscess. The lymphatic glands also may be enlarged. In the lymphadeno-leukæmia you may have white masses of lymphoid deposit in the liver, in the spleen, and in the kidneys. For a full account of the morbid histology of the case E. P— I will refer you to the 'Medico-Chir. Soc. Trans.,' vol. 83, 1900.

Digitized by Google

THE DIAGNOSIS OF THE CAUSE OF ACUTE ABDOMINAL PAIN.

A Clinical Lecture delivered at the Bristol General Hospital.

By CHARLES A. MORTON, F.R.C.S.,

Professor of Surgery in University College, Bristol; Surgeon to the Bristol General Hospital, and the Hospital for Children and Women.

GENTLEMEN,—The diagnosis of the cause of severe abdominal pain is of greater importance to the surgeon than to the physician, because he has to take the responsibility of deciding whether the abdomen shall be opened or not, and this is often a matter of very great difficulty indeed. It is true that there is hardly any risk in such a procedure if we are thorough in our antiseptic precautions; but it is a very distressing thing to a patient and his relatives to think he has to be operated on, and it means confinement to bed for some weeks. Then, again, although medical men understand that mistakes are inevitable at times, and that it may be less harmful to open the abdomen and find nothing abnormal than to leave a perforated gastric ulcer untreated for many hours, yet the public think we must be fools if we cannot say without a moment's hesitation exactly what is the cause of the abdominal pain; and if we open the abdomen and find nothing abnormal, the patient and his relations will probably consider that we ought never to have been allowed to practise. Yet every now and again the abdomen is opened for acute abdominal pain and nothing found there to account for it.

I think, therefore, we ought always to have the causes of acute abdominal pain clearly before our minds when we are called to such a case, whether as general practitioners or as surgeons. Possibly the general practitioner is most likely to err by not recognising the gravity of the case early enough, and the surgeon by thinking some condition needing immediate cœliotomy present when it is not.

In the first place we should consider whether the disease is really abdominal at all. Several cases of early acute mischief within the chest—pneumonia and pleurisy—have been operated on for an abdominal lesion, for in such cases the pain may be over the upper abdomen. The presence of cough, of rapidity of breathing out of proportion to the pulse, associated with high temperature, would be suggestive of the thoracic nature of the disease; as would also cyanosis, but cyanosis is a late rather than an early symptom. An examination of the chest should of course be made for physical signs of disease at one or other base. I have, on the other hand, seen a perforated typhoid ulcer simulate chest disease from the dyspnœa and cyanosis the condition of the peritoneal cavity produced, and I have had to operate on a perforated gastric ulcer too late, because the peritoneal friction was mistaken for pleuritic friction, and the abdominal pain, for pleuritic pain, before his admission to hospital. It is well to remember that friction at the base of the chest on the left side may be peritoneal and not pleural.

Then, again, with the disease of the spinal cord known as locomotor ataxia (or tabes dorsalis), severe attacks of pain in the abdomen and vomiting may occur—gastric crises, they are called. In such a case you may get the history that the patient has had several such attacks, and you may find an absence of knee-jerk, an ataxic gait, and pupil changes. I fear we shall be apt to forget this possible cause of abdominal pain unless the patient is known to have this disease.

In cases of severe abdominal pain, when associated with vomiting, always think of the possibility that a strangulated hernia may be present, and examine the hernial orifices. Do not be content to ask the patient if he has a hernia. The rupture may be so small he may never have noticed it.

I think it will be well, before considering the diagnosis of abdominal conditions which require operation, to bring before your notice several diseases which may cause very severe abdominal pain but do not require operation, or at any rate immediate operation. In simple colic, such as may occur after eating indigestible food, the pain may be severe, but is not usually accompanied by vomiting. In colic due to poisoning with lead or brass it may be very severe, and the paroxysms may continue for many days, and the abdomen may be hard and tender. Vomiting may also occur. The patient's work may suggest the condition, and in lead poisoning the blue line on the gums, and in brass poisoning the green line on the bases of the teeth, may point to these conditions. I

Digitized by Google

have been asked to explore the abdomen for a malignant growth of the colon, with a view to its removal, when the discovery of a blue line on the gums at once settled the diagnosis.

In the passage of gall-stones along the ducts the pain may be very severe, and may be almost anywhere in the upper abdomen. I have lately operated on a case in which it was always most marked in the right loin. Vomiting is often present, and may be very frequent. The history of previous attacks, especially if there has been slight jaundice after them, would be an aid in diagnosis, and in many cases the localisation of the pain to the region of the ducts and its extension through to the back would make the diagnosis almost certain. But there may be a very distinct history of such attacks, and yet the one from which the patient is suffering may be much more serious, for a large gall-stone may have passed from the gall-bladder directly into the duodenum by a process of ulceration and have blocked the small intestine. The persistence of the pain and vomiting in any case of supposed biliary colic should make us think of the possibility of this. In such a condition, however, there may be no history of previous attacks, for the whole gall-bladder may have been occupied by one very large stone. In some very rare cases it is thought a stone in the pancreatic duct may cause an attack very like biliary colic. In renal colic, due to the passage of a stone along the ureter, the pain may be abdominal rather than in the loin, and shooting into the groin or testis. Mr. Bowlby ('Brit. Med. Journ.,' July 1st, 1903) refers to such a case, and I have seen two cases in consultation as possibly intestinal obstruction which were probably renal colic. If morphine is given to relieve the pain, the constipation which follows—which in one of the cases would not yield to ordinary purgatives,—together with the abdominal pain, suggests intestinal obstruction. Vomiting, too, is often present in severe renal colic. Any tendency for the pain to start in the loin or to shoot into the groin or testis, any renal tenderness, and, of course, the presence of any blood in the urine, together with the history of the passage of gravel, or of similar attacks in which hæmaturia had been present, would be of value in the diagnosis from serious intra-peritoneal lesions.

There is another abnormal condition of the kidney which may lead to very severe attacks of abdominal pain, and that is too free mobility of the kidney, and this may cause pain in two ways. The pedicle of the too freely movable kidney may become twisted and the renal veins obstructed, leading to congestion of the kidney, and this may cause severe abdominal pain, with rigidity of the whole belly wall; or the too movable kidney may drag on the duodenum and cause severe attacks of epigastric pain and vomiting (Dietl crisis), so that if a very movable kidney is present, consider if the symptoms could be explained by the occurrence of one of these two conditions.

Embolism of the mesenteric vessels may produce abdominal pain, and vomiting and constipation, and thus simulate intestinal obstruction or peritonitis; and it is difficult to see how a differential diagnosis can be made. It is said by some that great tympanitic distension may occur in this condition, and this would be still further suggestive of some obstruction of the bowel, either mechanical or secondary to peritonitis.

Patients with acute inflammation of the pancreas have several times been operated on for intestinal obstruction; indeed, in most cases the nature of the disease has been discovered in this way. I have operated on one myself under the impression that intestinal obstruction was present. There is severe pain in the upper abdomen, frequent vomiting, and constipation. As you cut through the abdominal wall you will perhaps find the little areas of fat necrosis—little opaque white dots scattered about in the fatty tissue—which are so often present in this disease. I should not really refer to this disease in connection with diseases which do not require immediate operation, for the best thing we can do seems to be to get a good drain down to the pancreas, and a posterior drainage opening is useful as well; but even with such a proceeding the prognosis is very bad, and in nearly all the cases so far operated on the abdomen has been opened with the expectatation of finding some other condition present.

It is said* that acute osteomyelitis of the lumbar vertebræ may simulate peritonitis, or other acute affections of the abdominal viscera, by causing pain and distension of the abdomen. In such cases pyrexia and other symptoms of general constitutional disturbance would also be present. It is

* Makins and Abbott, 'Annals of Surgery,' 1896, vol. xxiii, p. 510.

Digitized by Google

thought that the abdominal symptoms may be due to irritation of the sympathetic ganglia and the nerve-roots in the abdomen. It would seem impossible to differentiate such a condition from acute visceral disease within the abdomen unless we can make out fixation of the spine, and tenderness and swelling on its posterior aspect. Probably vomiting would either be absent or little marked in this affection of the vertebræ.

We will now pass on to the consideration of the diagnosis of some abdominal diseases in which early operation is of the greatest possible importance. Perforated gastric ulcer is one of these. A sudden onset of any severe epigastric pain which persists and tends to spread over the abdomen, and is accompanied by collapse and tenderness over the upper abdomen, in a young anæmic woman with a history of attacks of gastric pain and vomiting, especially vomiting of blood, would be most suggestive of this condition. The pain is not invariably sudden in onset. I have lately operated on a case in which it was quite gradual in onset. I believe in this case the pain was at first due to localised peritonitis about the base of the ulcer, and that before adhesions had formed, actual perforation occurred. There may be free gas in the peritoneal cavity, but the gas does not always escape even when liquid does. The only sign of free gas in the peritoneal cavity before operation is an absence of liver dulness. But the liver dulness may be absent and yet there may be no free gas, for great distension of the colon with gas may obliterate the liver dulness. Therefore absence of liver dulness where there is no great general distension of the intestines is more likely to indicate free gas in the peritoneum than where general distension is great. But I have seen an absence of liver dulness in two cases without any such distension or any perforation of the stomach or intestines, and therefore it is not a sign we should place too much reliance on. In one case in which I was asked to operate for supposed perforation of a typhoid ulcer by the physician in charge of the case, the liver dulness was quite absent, though there was very little if any distension of the abdomen. I opened the abdomen and found that no perforation had taken place. In the other case there were acute abdominal symptoms, but the patient recovered without any operation. The position of the pain in perforated gastric ulcer may be misleading, for it is not always in the epigastrium. I have seen cases in which it has not been there, but at the side of the abdomen, in one case on the left and in the other on the right; and in one case it was in the epigastrium, but was just as much in the left iliac fossa—indeed, this was the region which the patient pointed to as the seat of pain. When the pain is in the right lower abdomen, appendicitis may be simulated; and such cases have been operated on for appendicitis and then the perforated gastric ulcer found. The gravitation of fluid from the stomach into the lower abdomen may, in some cases, explain this curious position of the pain. Although in some cases the shock at the time of perforation has been so severe as to cause sudden death, yet in others there is no collapse at all. In fact, patients may walk up to the hospital with a perforated gastric or duodenal ulcer, and you may find a pulse little, if at all, above 80. I remember one case of this kind which had been under treatment after perforation as a case of dyspepsia, and I did not get the chance of operating until general peritonitis had been well established. I have seen another case operated on too late because it was thought he had too good a pulse to have any serious abdominal lesion when he first came under observation. With regard to this I shall have something more to say when I come to speak of appendicitis.

It is well to remember that severe attacks of pain do occur in the course of gastric ulcer without evidence from the after progress of the case that perforation has been present. Possibly in such attacks perforation does occasionally occur when the stomach is empty and the opening becomes closed by adhesions, and in others a localised peritonitis is present around the base of the ulcer which has not perforated. A perforated ulcer may lead to a large abscess in the upper abdomen—subphrenic abscess—rather than to a general inflammation of the whole serous sac. Gastric ulcer is most common in anæmic young women, but may occur in elderly men without any anæmia; or a duodenal ulcer may perforate in an elderly man and cause symptoms exactly like those of perforated gastric ulcer. The abdomen has been opened by surgeons for perforated gastric ulcer in several cases, and no evidence of such found, and therefore—unless the case is a most typical one,—if we feel that the right thing is to explore the abdomen,

Digitized by Google

it is perhaps wiser to speak of the operation to the patient and friends as an exploration rather than as an operation for perforated gastric ulcer. And when the exploration is made it must be thorough. We must not think it is enough to just open the peritoneal cavity and see if there is any free gas, as I have seen recommended; for, as I have told you, again and again perforation has been found without free gas in the peritoneal cavity. The surface of the stomach must be carefully explored, and if nothing is seen on the anterior surface or the curves, then the posterior aspect must be explored through an opening into the lesser sac. Very often the sight of a red patch of inflamed peritoneum around the perforation directs us to it, or the hardness at the base of the ulcer, or sometimes on moving the stomach some gas or some liquid escapes from it. It is, of course, better in any suspicious but not quite typical cases of perforation of a gastric ulcer, to explore the abdomen than to run the risk of leaving a perforation too long unoperated on.

Severe abdominal pain may be due to acute intestinal obstruction, general peritonitis, or appendicitis. In appendicitis the pain is generally epigastric or umbilical at first, and later becomes localised in the appendix region, and at the commencement of the illness it may be impossible to distinguish between these three diseases. The hard, board-like abdominal wall and extreme tenderness, with greatly deficient abdominal respiratory movement, would be suggestive of an inflammatory lesion rather than a mechanical obstruction; and if the contraction of the abdominal muscles and the tenderness were general, of a general peritonitis rather than a localised one such as appendicitis. Very early great distension would be certainly suggestive of volvulus. In other forms of mechanical obstruction, and in all forms of peritonitis, distension would not be present to a marked extent at the very onset of the pain. The passage of blood and mucus, especially in a child, would be suggestive of intussusception. The bowels may fail to discharge either fæces or flatus for many hours in health, and therefore the "obstruction" symptoms are of no value in diagnosis when the pain begins, nor the visible or palpable contraction of coils which is so valuable a means of differentiating mechanical obstruction from the paralytic obstruction of peritonitis at a later stage. Remember diarrhœa is not rarely one of the early symptoms of appendicitis, and also sometimes of pneumococcal peritonitis, and that in pelvic peritonitis rectal tenesmus with discharge of mucus is not rarely present. Do not lay stress on the absence of the so-called small wiry pulse of peritonitis; it may be a very good pulse at the commencement of the disease—neither small nor rapid. In peritonitis or acute intestinal obstruction one should certainly expect to find vomiting present, as well as severe pain, at the onset; and in cases of well-localised appendicitis vomiting as well as pain is often an early symptom. Do not, however, be misled by the absence of vomiting. I have had under my care several cases of peritonitis in the lower abdomen from disease of the appendix—cases in which there has been free pus in the lower abdomen and in the pelvis, and lymph on the coils of intestine,—and yet there has been no vomiting, though the patient took liquids by the mouth. In acute abdominal disease we must, in making a diagnosis, depend more on the symptoms and signs that are marked than on the absence of certain symptoms and signs which we might expect to be present. I opened the abdomen of a man the other day and found malignant stricture of the sigmoid, enormous distension of the intestines, perforation of a stercoral ulcer, and free gas in the peritoneum, and general peritonitis; and yet he had only vomited once since the onset of the severe pain which marked the perforation, twenty-four hours before I operated; and he passed flatus freely within an hour of the operation without any injection into the bowel.

If abdominal pain is due to appendicitis, though it is often in the epigastric or the umbilical region, or even over the whole abdomen at first, it soon becomes localised to the reigion of the right lower abdomen, unless the appendix has been hanging over into the pelvis and perforated there and then, it may never be present more in the right side of the abdomen than elsewhere in the lower half. Occasionally the appendix causes suppuration on the left side of the abdomen. I have operated on one case of this kind in which the appendix lay across the top of the sacrum, and around the tip on the left side was an abscess. And not only does the pain in appendicitis tend to become concentrated in the area where the appendix lies, but a swelling forms there in many cases. If such

Digitized by Google

a swelling cannot be felt through the abdominal wall, it may be felt *per rectum;* and if no swelling can be felt *per rectum*, marked tenderness on pressing on the structures around with the finger in the rectum may be a valuable indication of pelvic appendicular peritonitis—for tenderness in such a condition may be much greater on pressure on the inflamed peritoneum through only the thin rectal wall than from pressure through the abdominal wall. In well-localised appendicitis we should expect some rise of temperature, but in general infection of the peritoneum from perforation or gangrene of the appendix this may be quite absent. The great difficulty is to say in any severe case of appendicitis that the mischief is well localised, and has not already spread widely in the lower abdomen and pelvis. In cases in which it is thus spreading there may be no vomiting, and a pulse under 100, and the temperature very little raised. The persistently severe pain without definite swelling, the marked tenderness over the lower abdomen and *per rectum*, with perhaps marked limitation of abdominal breathing, and some rigidity of the lower belly wall, are the suspicious symptoms and signs.

I have not as yet referred to the pelvic peritonitis of women due to inflammation extending from the Fallopian tubes, which generally remains limited to the lower abdomen. The pain is frequently in the lower abdomen, frequent and painful micturition is a common symptom, and the temperature is often raised. At first marked tenderness, and later swelling, is detected *per vaginam*. We must, however, remember that pelvic peritonitis in a woman, as well as in a man, may be one form of appendicitis; and the frequent and painful micturition of perimetric inflammation may be present in a marked degree in the pelvic form of appendicitis in the male; and I have known a very severe pain shooting into the rectum and anus present in this condition also. In pelvic inflammation in women (not apparently connected with the appendix) which has subsided, I have found the pain at the onset so high up in the abdomen as to suggest a more general and serious form of peritonitis.

There is one other condition attended by severe abdomina pain, the diagnosis of which is of very great importance, because early operation is generally so imperative and so successful. I refer to tubal pregnancy. If a woman is seized with very severe abdominal pain and gets very pale, possibly vomits, and the abdomen becomes somewhat distended and rigid, with shifting dulness in the flanks, always suspect this condition. You will very likely get a history that she has just aborted, but this supposed abortion may have been only the casting off of the uterine decidua accompanied by hæmorrhage. Such a condition is common before the pregnant tube ruptures. Then you may find that the woman missed a period before that. The pain and collapse might be present with any perforation into the abdomen, but the sudden onset of great pallor, and shifting dulness in the abdomen due to extravasation of blood, together with the history mentioned, would probably enable a correct diagnosis to be made.

Mr. Rutherford Morison, of Newcastle, says* that in several cases he has known the abdomen opened for acute disease, such as intestinal obstruction or peritonitis, and nothing found at the operation or post mortem but contracted granular kidney. He thinks such cases may be distinguished by the aspect of the patient and the condition of the urine. I have had no experience of such a condition, and it seems to me it would be very difficult to say in any case that Bright's disease was the only disease present, for we must remember that various acute abdominal conditions demanding early operative treatment may be present with Bright's disease; and although the complications of Bright's disease add greatly to the gravity of the prognosis after any operation, one would hardly be right in not giving them the only chance of recovery if it lay in operation. If a patient had strangulation of the intestine, surely we should be wise in operating for his relief even if he had granular contracted kidneys.

* 'Lancet,' January 31st, 1903.

Jacques and Parizeau report the case of a boy of eleven brought to the hospital suffering from vomiting and tympanitis, accompanied by pain. Laparotomy was performed, and the seat of obstruction was found to be at the sigmoid flexure. An indurated mass was removed, but the child died of shock. The neoplasm was found to be a typical adeno-carcinoma. The cancer developed insidiously, giving absolutely no premonitory symptoms except chronic dyspepsia, but causing a sudden occlusion of the intestines.—*American Journ. of Obstet.*, December, 1903

Digitized by Google

A DEMONSTRATION OF CASES AT THE MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

By BYROM BRAMWELL, M.D., F.R.C.P.E.

GENTLEMEN,—I come before you with considerable diffidence, because, not knowing anything about the cases which we are to see, I may not be able to say much that may be interesting to you. But I will do my best.

CASE. 1. *Chronic cough and spit in a girl æt. 7.*—The first case is this little girl, æt 7. When a year old she had, I understand, an attack of bronchitis followed by pneumonia; and since then she has been almost continuously troubled with a cough and spit. She is, as you see, a delicate-looking, pale, thin girl. Her chest presents considerable alterations; there is very marked flattening and contraction at the bottom of the thorax on each side; the expansion is deficient. On percussion there is some impairment of the note, more particularly at the upper part of the right lung; the note is not very good over the upper part of the left lung. On auscultation I find numerous fine crepitations over the greater part of each lung. The expectoration is said to be copious in amount, thick, and yellow; it is brought up in nummulated masses. The question, of course, seems to be, what is the nature of the lesion? She has never spat blood, and she does not get unduly hot or perspire at night. The other children of the family are quite well; the mother and father are healthy. The mother lost one brother by consumption, but no other member of the family has had chest trouble. So far as I have been able make out in the hurried examination which I have just made of the chest, there is no definite evidence of any localised excavation, of a cavity; but the gentleman who kindly brought the case to us has, I understand, detected evidence of a cavity about the middle part of the right lung behind. Now what is the nature of the condition? Three things in particular suggest themselves to my mind. One is phthisis, another is merely chronic bronchitis, and a third is bronchiectasis. If one were quite satisfied that there is definite evidence of excavation, then of course the diagnosis would lie chiefly between phthisis and bronchiectasis. The position of the cavity in the middle of the lung is of course strongly in favour of bronchiectasis. The child, however, is very young for bronchiectasis, but the same statement also applies to phthisis. One most important point in the differential diagnosis of these two conditions is the examination of the sputum for tubercle bacilli. This has, I understand, been done on more than one occasion, but without any bacilli being found. That is a point of very considerable practical importance, although, of course, everybody knows that the absence of tubercle bacilli does not definitely exclude tubercle. The sputum is not brought up in large quantities at a time, and it is not fœtid; these characters are certainly against an ordinary bronchiectasis. But the sputum is brought up in nummulated masses; that is suggestive of a cavity. My own feeling, from what I have seen of the case, would be rather against bronchiectasis, I must confess; but it is, of course, exceedingly difficult, after examining a case like this for a few minutes only, to give a final definite—a dogmatic—opinion. The gentleman who has had the patient under observation during the whole of her life, and who has had repeated opportunities of examining the chest and considering the diagnosis, is very much more able to express an opinion on the case than I am. He thinks, I understand, that there is bronchiectasis. I simply say that so far as I can judge from the hurried examination which I have just made, I should hesitate to give a definite diagnosis of bronchiectasis.

With regard to treatment, which is of course a a most important point in a case like this, one must endeavour to maintain the general health in the best possible state of efficiency; to see that the patient has plenty of fresh air, and, if possible, sunlight—a very difficult thing at any time in this climate of ours, such as it has been during the last year; to feed her up; to give her plenty of milk, cod-liver oil, etc. It is also very important to endeavour to protect her against recurring attacks of bronchitis. We are told by the mother that whenever the child gets cold it flies to her chest at once, and she gets worse. She should be given tonics, such as quinine, nux vomica, syrup of the phosphates, and so on. In cases of bronchiectasis I have been impressed with the beneficial effects of intra-laryngeal injections of menthol. Of course, if

Digitized by Google

in a case of bronchiectasis the cavity is large and is so situated that it can be reached by the surgeon, operative procedure is no doubt often very beneficial; but it is not always easy to find or reach the cavity. In some cases of bronchiectasis in which I thought I had exactly localised the cavity, no cavity was found by the surgeon. The cavity may of course be too deeply situated, or the incision may not have been made over the right place. In a recent case of this kind in which there was without doubt a large bronchiectatic cavity, in which large quantities of fœtid expectoration were brought up at intervals of a few hours, and in which the auscultatory signs of a cavity seemed quite distinct, no cavity was found at the operation. In that case a remarkable improvement resulted from the intra-laryngeal injections of a 5 per cent. solution of menthol in olive oil. In children it is difficult to carry out the intra-laryngeal injections, but I find that spraying the throat with the same solution and allowing the child to breathe the spray well into the lungs answers the purpose in many cases extremely well. In phthisis, too, intra-laryngeal injections of menthol are certainly often very beneficial.

Case 2. *Tabes with Charcot's joint lesion.*—The next patient I have not examined, but he brings a written statement of his chief symptoms. He is æt. 44 years, has been married twenty years, and his occupation is that of a traveller for clocks. He complains of shooting pains in his legs. He has had these pains for ten or twelve years. They do not come on at any particular time. He states that before an attack of pains he is usually extra well; that is of some interest—I don't remember having heard any of my tabetic patients make such a statement. He thinks that changes in the weather have some influence in producing the attacks. That is, of course, very common; the pains of tabes are often induced by changeable, wet, windy, stormy weather. In some cases alcohol excites the pains. This man has not noticed that; on the contrary, he thinks that a little Scotch whisky relieves the pains. A gentleman recently consulted me for tabes, and he made the very definite statement that if he took a glass of beer or a glass or two of wine he was sure to have an attack of pain. I asked him if he was gouty, and he said he was; and he then volunteered the very interesting statement that if he took a few doses of piperazin—an anti-gout remedy—he could relieve the pains. It is an important practical hint. It seems to show that in some gouty people the pains of tabes are made worse by alcohol, and that anti-gout remedies may in such persons relieve the pains. I have not tried piperazin for the relief of the lightning pains of tabes, but I intend to do so. Phenacetin is the remedy which I usually prescribe, in the first instance at all events—twenty grains of phenacetin, repeated if necessary two or three times within a few hours. If phenacetin or antipyrin do not give relief, I usually employ morphia. This man has had pains in his legs, but nowhere else. We find in a large number of cases of tabes that the pains are confined to the legs; but I need not say that in some cases the trunk and arms are affected. The lightning pains are rarely felt in the head. The patient complains of a swelling of the right foot. The swelling is very obvious, even through his boot; no doubt we shall find that it is an example of Charcot's joint disease. He says that he has no difficulty in making water, though at one time it used to dribble away. His bowels are, he says, quite regular. His appetite and digestion are good, and he has had no attacks of vomiting. On looking at him one sees that his pupils are extremely small; the myotic condition of the pupils is a very characteristic feature of many cases of tabes. One can often, from the facial physiognomy alone, and without asking the patient any question, come to the conclusion that he is probably suffering from tabes; a sallow, somewhat haggard expression of countenance, with pin-point pupils, is very suggestive of tabes. General paralysis of the insane is another condition in which the same physiognomy may be present; but it is more characteristic of tabes. Of course opium has to be excluded. Judging from the movements which the patient has made since he came into the room, there does not seem to be any marked degree of ataxia. He walks, you will see, with a slightly unsteady gait, but that, he thinks, is largely due to the condition of his foot. There is some Rombergism, but it is not very marked. The finger-nose test is almost perfectly performed, showing that there is little or no inco-ordination in the upper extremities. The leg below the knee is very much swollen; this is due to a thickening of the soft parts; but the bones of the ankle and foot seem

Digitized by Google

to be much thickened. The sole of the foot is filled up; it looks very flat. The patient states that the instep began to swell a year ago, but that the swelling of the leg was not noticed until July last. I have seen several cases of Charcot's joint disease affecting the ankle and foot; the swollen condition at the inner side of the ankle, and the flat, filled-up appearance of the sole, are very characteristic. I do not remember to have seen such an extensive solid œdema of the soft parts as is present in this case, in any of the cases of Charcot's disease of the foot which have come under my notice. The solid œdema of the soft parts reminds one of obstruction of the lymphatics, of elephantiasis; a similar condition is sometimes seen associated with repeated attacks of erysipelas. The patient says the leg is not painful except when he gets the lightning pains. There can, of course, be no doubt that this patient is suffering from tabes. But in many cases which come before us, especially when there is no ataxia, one has to look for the physical signs which characterise the disease. The condition of the reflexes, the condition of the pupils, and the presence or absence of thoracic and ulnar analgesia, are important points. In this case the knee-jerks are, you will see, absent even on reinforcement. The Achillis-jerks, too, are absent; owing to the great œdema of the right foot the Achillis-jerk on that side cannot be properly tested, but the jerk is, you will see, quite absent on the other side. The best posture for getting the Achillis-jerk is to make the patient kneel on a chair. You will notice that the big toe-nail on one side is wanting. The patient states that the nails of the big and second toes have several times fallen off. The shedding of the nails is, he says, preceded by discoloration—hæmorrhage—below the nail before it comes off. This is a common and very characteristic trophic lesion in tabes. The plantar reflex, to which I attach so much importance as a sign of organic disease—as, for example, in the early stages of disseminated sclerosis—is usually normal in cases of tabes. In testing the plantar reflex it is important to relax the knee; the patient should be made to lie on his side with the foot in this position and the toes relaxed; the sole should then be stroked with a blunt instrument, such as the end of a tuning-fork, or with the finger-nail, and the reflex movements which result in the toes, foot, thigh, etc., should be carefully noted. The most important point is the movement of the big toe. In tabes the toe movement, if any, is plantar flexion; extension (dorsi-flexion of the big toe) is very rarely observed in tabes. In the few cases in which I have seen an extensor response in tabes there has generally been some explanation, some associated lesion. In one of my cases of tabes, for example, in which a double extensor response was present, the history showed that the patient had had two successive attacks of hemiplegia some years before, one on the right and one on the left side; the double extensor response was the remains of these hemiplegic attacks. In this particular case, then, the knee-jerks and Achillis-jerks are absent; there are lightning pains; the pupils are myotic, and do not react to light. Let me say that when the pupils are so markedly contracted as they are in this case it is very difficult to be sure of the contraction of the pupil to light; under such circumstances I generally find that by using an electric light, which can be suddenly turned on, and by magnifying the pupils with an ordinary lens, such as one uses for the indirect ophthalmoscopic examination, one can often determine whether a very small pupil contracts to light or not, when it is difficult to be sure of the contraction if the pupil is examined in the ordinary way. The patient states that his sight is good, and the optic discs are said to be normal. The patient states that his sexual power has been failing lately; this, of course, is very common in tabes. In all cases of tabes it is an important point, more especially from an etiological and scientific point of view—for unfortunately a history of syphilis has not in most cases of this disease any very material bearing upon treatment,—to ascertain whether the patient has had syphilis or not. As everybody knows, syphilis is usually believed to be the most important cause of tabes, just as it is the most important cause of general paralysis of the insane. I agree with the opinion. In the great majority of cases of tabes the patients have had syphilis. This is a case in point; this patient had syphilis twenty-five years ago. Of course one not infrequently meets with cases in which syphilis is denied, but I need not say that a denial of syphilis does not count for very much. Still, there are cases in which, so far as one can ascertain, the patient is perfectly straightforward when he denies having had syphilis.

Digitized by Google

There can, I think, be no question that some patients who have had syphilis are not aware of the fact. It is said that in 10 per cent. of the cases in which an undoubtedly syphilitic skin eruption is present, it is impossible to obtain a history of a chancre, or to detect any appearances indicative of a primary sore. But I need not say that in many of the cases in which tabetic patients deny that they have had syphilis, they have had the disease, and they know that they have had the disease. In this case there does not seem to be any thoracic or ulnar analgesia. One other point, regarding the eyes. You will see that the left eyelid drops; the patient himself has noticed this. There seems to be a certain amount of ptosis; that is a common condition in tabes. This patient has no pain in his back. He does not think that the disease is progressing much. The case is a highly characteristic one. The chief point of interest is the condition of the foot, the Charcot's joint disease, with a great deal of solid œdema of the leg—the sort of œdema which one gets as the result of lymphatic obstruction, the sort of solid œdema of the soft parts which is present in elephantiasis. As I have already said, I do not remember to have seen such a marked degree of solid œdema in any case of tabes before. I need not say anything further about this case.

(*To be concluded.*)

Treatment of Eclampsia.—According to Bumm ('Münch. med. Woch.,' No. 21), 43 cases of eclampsia treated by morphine, chloral, chloroform, diaphoresis, with bleeding and transfusion in a few cases, gave a mortality of 30 per cent. In contrast he has since treated 25 cases by immediate delivery by abdominal or vaginal section, forceps, or version, with a mortality of 12 per cent. He favours the latter treatment, especially when the patient is seen soon after the first convulsion, with pulse and lungs in good condition. If the cervix is well dilated he would employ forceps or version, according to the position of the head, and perforation if the child is dead or dying. If the cervix is dilated just enough to permit combined version, he would perform this and hasten dilatation by tractions upon the foot brought down. If the cervix is not dilated and the woman's condition is such as to demand immediate delivery, he would favour Dührssen's vaginal Cesarean section rather than risk the use of metallic dilators.—*American Journ. of Obstet.*

Endovenous Injections of Sublimate.—Maragliano states that the experience at his clinic has shown that these injections can be made without the slightest inconveniences if a large vein is selected for them, and they are made in such a way that the blood continues to flow through the vessel and thus mixes continually with the fluid injected. The solution must be weak, 1 to 10,000 or 1 to 5000, or even 1 to 1000 is harmless if the above rules are strictly followed. He injected from 1 to 5 mgm. of sublimate a day in progressing doses in man, examining the blood from day to day. No changes could be detected in the blood except a slight leucocytosis. From experimental research on animals he concludes that the minimal toxic dose is 15 mgm. per kilogram in a rabbit. Intoxication with sublimate has a tendency to destroy the blood-corpuscles, but this has nothing in common with the therapeutic doses of sublimate. The slow minimal therapeutic intoxication induces a leucocytosis which probably generates immunising substances active against both bacteria and toxins. This assumption harmonises perfectly with what is observed after endovenous injections of sublimate in certain infectious diseases. They unmistakably increased the resisting powers of animals injected, conferred agglutinating power on the serum and preserved the animals against toxic doses of bacterial toxins. He succeeded in saving the animals from the results of experimental diplococcus and streptococcus peritonitis and from anthrax by endovenous injection of $\frac{1}{50}$ mgm. of sublimate per kilogram in a weak solution. This result was obtained both by preliminary injections of small repeated doses, as well as by postponing the injections until after the infection. The researches reported establish the efficacy of small doses, and that the action of the drug is due not to its bactericidal power, but to the increase in the organic resistance and the production of antitoxic material. Other drugs were tested in endovenous injections, and the dosage found for tincture of iodin, antipyrin, Fowler's solution, etc. One patient with rebellious hemoptysis was treated by endovenous injection of 150 c.c. of a 2 per cent. solution of gelatin, which arrested the hæmorrhage almost instantaneously. These researches were conducted mostly by Mariani, and included much experience with endovenous injection of oxygen, demonstrating its efficacy in certain conditions and its harmlessness.—*Journ. A.M.A.*, January 2nd, 1904.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 588. **WEDNESDAY, FEBRUARY 3, 1904.** **Vol. XXIII. No. 16.**

CONTENTS.

PAGE

***On Injuries and Diseases of the Orbit.** By E. TREACHER COLLINS, F.R.C.S. **241**

***A Lecture on Some Points in Balneary Therapeutics.** By LEONARD WILLIAMS, M.D., M.R.C.P. **249**

***A Demonstration of Cases at the Medical Graduates' College and Polyclinic.** By BYROM BRAMWELL, M.D., F.R.C.P.E. *(concluded)* **254**

** Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

ON INJURIES AND DISEASES OF THE ORBIT.

By E. TREACHER COLLINS, F.R.C.S.,
Surgeon Royal London Ophthalmic Hospital; Ophthalmic Surgeon Charing Cross Hospital.

GENTLEMEN,—In proceeding to give you a brief account of my clinical experience of injuries and diseases of the orbit, I think it will be best for me first to speak generally of the symptoms to which they give rise and the way to investigate them.

The commonest of all symptoms is some displacement of the eyeball. It may be protruded forwards or sunk backwards in the orbit, the former condition being spoken of as exophthalmos or proptosis, and the latter as enophthalmos. Each of these symptoms may be brought about in a variety of ways.

Exophthalmos may be due to—

(*a*) The presence of some extraneous substance in the orbit (new growth, inflammatory exudate, blood, or air).

(*b*) The encroachment on the cavity of the orbit of some growth external to it, or of some distension of one of the neighbouring sinuses.

(*c*) The dilatation of the blood-vessels in the orbit, as in Graves' disease.

(*d*) The contraction of the unstriated muscle-fibres of Müller situated at the back of the orbit about the spheno-maxillary fissure. These are supplied by the cervical sympathetic nerve, stimulation of which results in slight proptosis. "The starting of the eyes from the head" caused by strong emotions, which writers of fiction are so fond of describing, is probably produced in this way.

Enophthalmos may be due to—

(*a*) Absorption of fat, or atrophy of the tissues of the orbit.

(*b*) The formation and contraction of cicatricial tissue in the orbit.

Digitized by Google

(*c*) Fracture of the orbit, and the escape of some of its contents into the antrum of Highmore.

(*d*) Paralysis of the muscle-fibres of Müller above referred to.

(*e*) Possibly to rupture or faulty development of the check ligaments which normally prevent the recti muscles on contraction drawing the eyeball backwards.

In the majority of cases displacement of the eyeball backwards or forwards is readily detected, but cases are sometimes met with where the displacement is so slight that it can only be made out after the careful comparison of the two sides.

In these doubtful cases I think it is best for the patient to be seated in a chair, the surgeon standing behind raising the two upper lids and looking down from above, the patient at the same time being directed to look at the floor.

Another useful way of conducting the examination is to place the edge of a card in front of the eye, letting it rest against the brow and the cheek; the distance of the cornea from it can then be noted, and compared with the distance of the cornea from a card on the other side when held in a similar way.

Besides being more or less protuberant in the orbit, the eyeball may be displaced laterally or vertically. In a case of orbital growth the direction in which the eye is displaced helps considerably in determining its nature. Thus in a tumour starting in or around the optic nerve there is usually exophthalmos, without any lateral or vertical displacement. In a case of distended frontal sinus the eyeball becomes displaced downwards and outwards. A tumour originating in the lachrymal gland, on the other hand, displaces the eye down and in; whilst a growth starting in the antrum pushes the eye upwards.

Limitation in the movements of the eye, which is often met with in affections of the orbit, may be caused mechanically, by the presence of some adventitious substance, or by implication of one of the extra-ocular muscles or of the nerves supplying them.

The presence of some limitation of movement is often suggested by the patient complaining of double vision. It can usually be easily detected by comparing the amount of movement of the eyes on the two sides when the patient tries to look in different directions.

In affections of the orbit much can usually be learnt by palpation. The presence of a fracture or irregularity in its bony margin may be detected. The presence of any abnormally sensitive spot may be located. If air has passed into the cellular tissue beneath the skin of the lids, the peculiar crackling sensation of emphysema can be made out. If some abnormal swelling is encountered its consistency may be estimated. It should be determined whether it is nodular or smooth, whether it is elastic or bony hard, whether it pulsates or fluctuates. Some cases of distended frontal sinus present a characteristic sensation like egg-shell crackling.

The readiness with which the eyeball yields on pressure backwards of it in the orbit, through the closed lids, gives some indication as to the presence or absence of some solid growth behind it.

When pulsation of an orbital swelling has been detected auscultation should be resorted to, to find out if a bruit can be heard. The bruit of arterio-venous aneurysm of the orbit is usually audible to the patient. In a case of this sort which I had under my care some years ago, the constant buzzing noise was the chief cause of the patient's complaint. It occasioned him excessive annoyance, and so loud was it that he said his wife heard it at night.

In all orbital affections the appearances of the eye are of importance in affording evidence of pressure or obstructed circulation. The vision and refraction should be tested on the affected side and compared with that of the fellow eye.

In cases of orbital growth the globe may become so pressed upon as to become somewhat flattened. There is a specimen of an eye which I mounted in the Moorfields Hospital Museum, where one side has been so compressed by an orbital growth that the outline of the globe on antero-posterior section is triangular in shape.

In a case of tumour of the optic nerve which I removed, the presence of gradually increasing hypermetropia materially assisted me in arriving at a correct diagnosis before operating on the patient. In another case of orbital tumour, a sarcoma, the presence of hypermetropia on the affected side, and the absence of any refractive error on the other suggested pressure on the eye from without. Obstructed circulation of the ocular blood-vessels may give rise to external congestion, subconjunctival hæmorrhages, or chemosis

Digitized by Google

of the conjunctiva. Ophthalmoscopically the optic disc may be found swollen or atrophied, and the retinal vessels enlarged or narrowed as the result of pressure.

In some cases of proptosis of the eye the most careful investigation of the symptoms leaves it uncertain if a growth is or is not present in the orbit; or if it has been determined that a new growth is present, it is doubtful whether or not it is of a malignant character. Under such circumstances it is sometimes found advisable to perform an exploratory operation on the orbit.

Where a new growth can be felt, and it is its nature which has to be determined, an incision must be made directly down upon it and a piece cut out for microscopical examination. As an example of how even this test may prove fallacious as regards the nature of the tumour, I may mention an incident which happened to me while pathologist at Moorfields Hospital.

There was a patient with a swelling in the orbit of doubtful nature. The surgeon cut down and removed a piece of tissue, which was sent to me for examination. I cut sections of it, examination of which with the microscope showed it to be composed of fibrous tissue with round-celled infiltration. I reported that the specimen was apparently a fibro-sarcoma. As there was still a mass in the orbit the surgeon performed a second operation, and on cutting down this time came upon an unmistakable dermoid cyst. What he had removed at the first operation and sent to me must have been a piece of inflamed fibrous tissue of the orbit external to the cyst.

Where no tumour is to be felt, and it is considered advisable to explore the orbit, it is best to cut down through the skin and subcutaneous tissue on to its outer margin. Then, after separation of the palpebral ligament from the bone, the finger can be introduced into the orbit and passed, if necessary, right back to its apex. As the outer margin of the orbit shelves away the finger is more easily introduced from this position than elsewhere. An incision in this position, moreover, does not interfere with the movements of the eyelids.

An operation has of recent years been introduced by which not only may the orbit be most effectually explored, but by which the structures behind the eye can be exposed to view. It is named Krönlein's operation, after the surgeon who first introduced it. I have performed it once myself for the removal of an orbital sarcoma with a most satisfactory result. As I do not think there are many cases in this country in which it has been employed, I will give you a description of my case:

Edward L—, æt. 7, attended at Moorfields Hospital on September 3rd, 1900. What was taken to be a cyst was dissected out of his right orbit. Microscopical examination of the tissue removed showed it to be sarcomatous. He was readmitted into the hospital on March 6th, 1901. The right eyeball was proptosed; its movements were good. A firm mass could be felt in the orbit somewhat above and to the inner side of the globe. Externally the eye appeared healthy. Ophthalmoscopically the edges of the optic disc were seen to be slightly blurred. V. of R. = $\frac{6}{18}$ c̄ + 1·5 D. $\frac{6}{6}$; and V. of L. = $\frac{6}{6}$.

The following day, the child having been anæsthetised, I made a slightly curved incision along the outer margin of the orbit right down to the bone, beginning above at the anterior end of the temporal ridge, and ending below at the upper border of the zygomatic arch. The palpebral ligament and the periosteum attached to the outer margin of the orbit were then divided. The latter, together with the contents of the orbit, were drawn inwards. A wedge-shaped piece of the outer bony wall of the orbit was next cut through with a chisel, the apex of the wedge being backwards. This wedge-shaped piece of bone, with the soft tissue attached to its external surface, was drawn outwards. After the periosteum had been cut through the orbital contents were then exposed to view. By a little separation of the tissues I came down upon the surface of the tumour, which appeared to be encapsuled. With my finger I was able to separate it easily from all surrounding structures except anteriorly, where there was a band of adhesion, probably corresponding to the position of the previous operation. This band had to be cut through with scissors.

After the removal of the tumour the wedge-shaped piece of bone was restored to its proper position, the skin incision united with sutures, and an antiseptic dressing applied.

This dressing was not removed until four days later, when the wound was found to be flat and healing well. The boy made an uninterrupted recovery. A fortnight after the operation the vision

Digitized by Google

of the right eye was = $\frac{6}{24}$ + 1·5, $\frac{6}{24}$ J.1 partly. The movements of the eye in all directions were somewhat limited, especially outwards. The pupil of the right eye was larger than that of the left, and acted but sluggishly to light. Subsequently he regained good movements in the eye in all directions except outwards. I have not seen him for some time, as he lives at a distance in the country, but I had a letter from his mother on April 27th of last year—that is, two years after the operation—saying the boy was quite well and made no complaint of his eye.

Hæmorrhage into the orbit may result from injuries to it produced either by direct or indirect violence. Hæmorrhage into the orbit following an indirect injury usually means a fracture of the anterior fossa of the base of the skull.

I was recently asked to see such a case at the Charing Cross Hospital in which the hæmorrhage had produced the most extreme proptosis. So great was it that the lids could not be closed over the surface of the cornea. The extreme tension of the parts had abolished all power of movement in the eye. The conjunctiva between the lids was congested and chemosed. The surface of the cornea, from exposure, was dull and xerotic, showing every sign of shortly becoming ulcerated.

As a rule hæmorrhage into the orbit does not call for any interference; the blood absorbs, and the eyeball returns to its normal position. In such an extreme case as that I have just mentioned, if nothing were done the sight would certainly be lost from ulceration of the cornea.

It would therefore be well to make an incision along the outer margin of the orbit and turn out the blood-clots, the margins of the lids afterwards being stitched together over the surface of the eye. The most strict asepsis would of course have to be ensured to prevent the occurrence of any cellulitis.

When a fracture of the orbit extends into one of the air sinuses in its vicinity, such as the ethmoidal, air may become forced into the cellular tissue of the orbit, producing emphysema. The following case admitted to Moorfields Hospital on April 11th, 1900, is a good illustration of this. William B—, æt. 17, when boxing the previous morning, was struck in the right eye; his nose bled a little and there was slight pain, which passed off. Several hours later the eye began to become more prominent than its fellow, and pain of a much severer character set in. On examination the right eyeball was found considerably proptosed. The skin of the lids was tightly stretched, red, and shining; on palpation of them a faint sense of emphysematous crackling was experienced. There was some difficulty in separating them to gain a view of the eyeball; the latter appeared healthy, but its movements in all directions were somewhat restricted. When the patient made a violent expiratory effort with the nostrils closed, the amount of proptosis markedly increased; afterwards, however, the eyeball could be pushed back into nearly its former position.

The patient was cautioned against coughing or making any expiratory efforts, and in nine days' time he left the hospital, the distension of the lids having subsided and the right eyeball being but very slightly more prominent than the left.

We meet with cases of orbital cellulitis sometimes going on to the formation of an abscess which points in the lid, and where the history is elicited of some direct injury without wound previous to the onset of the trouble.

At first, without any wound, it seems difficult to account for the suppurative inflammation in the orbit. I think, however, the explanation in such cases is that the blow causes fracture of one of the thin-walled air sinuses into the orbit, and that infection gains entrance from the air in them.

In the following case, which ended fatally, and which I had to do with when a house surgeon, that is probably how infection occurred.

A lad æt. 17 came and stated that a month ago he received a blow on his left eye from a turnip; the eyelids were for a time swollen and much discoloured, but afterwards resumed their normal appearance. Lately, swelling of the upper lid had commenced again. An abscess was found to be pointing; an incision was made into the centre of the upper lid, pus escaped, and a drainage-tube was inserted. Two days later the swelling of the orbital tissue had considerably increased, and his temperature was 101°. Two days later still it rose to as high as 107°. The incision in the orbit had been enlarged, and the wound was being syringed out with a perchloride of mercury lotion. He had repeated rigors; abscesses formed, and were opened, over the parietal bone and at the inner side of the nose. He

Digitized by Google

became unconscious, and died comatosed ten days after he first came under treatment.

At the post-mortem examination extensive purulent meningitis was found, and an abscess in the left frontal lobe. There was some necrosis of the bone forming the roof of the left orbit, an abscess cavity between the periosteum and the bone, and pus in the frontal and ethmoidal sinuses. The left cavernous sinus was filled with semi-solid purulent material.

Fractures involving the orbit either from direct or indirect violence sometimes pass through the optic foramen. The optic nerve may then be wounded, and the patient find after the injury that the sight on that side has become completely destroyed.

I saw a case of this sort in a man who received a severe blow on the temple in a fall from a gig. He was unconscious for a short time, but on recovering found the eye on the injured side was quite blind. Ophthalmoscopic examination some days after the injury showed no changes. The optic disc was of a good colour, and the retinal vessels were of normal size. As the injury to the nerve was behind the position where the central artery enters it, the vessels derived from that artery remained unaffected. Some months later, when sufficient time had elapsed for a descending atrophy of the nerve to manifest itself, the optic disc was found to have become pale in colour.

Sudden loss of sight may similarly come on from gunshot injuries of the orbit, probably from direct wounds of the nerve.

In February, 1895, I saw a man æt. 53, who while out rabbit shooting a month previously was struck by some pellets in his right orbit. He told me that immediately afterwards he shut his left eye and found that the sight of the right had nearly gone, and that five minutes afterwards it had quite gone. Two scars in the neighbourhood of the right brow could be seen, one just above it, beneath which a shot could be felt. The other, at which probably the shot had entered which injured the nerve, was just below the orbital margin. He was unable to tell light from dark with the eye, and ophthalmoscopically there was some pallor of the outer half of the optic disc, but no other changes. I saw him again a year later, and the amount of pallor of the disc had considerably increased, but the retinal vessels retained their normal size.

I have had three patients who have been shot through the head, the bullet in each case passing through the two orbits and producing blindness of both eyes. "Having a bullet through the head" is generally regarded as synonymous with death, but all these three patients survived. The first case was a very sad one. A man æt. 34, who was deaf and dumb from scarlet fever, attempted to commit suicide by firing a revolver held against his right temple. The bullet emerged at the left cheek, just below the margin of the left orbit. The sight of the right eye was at once destroyed, and the eyeball, which must have been wounded posteriorly, became shrunken and puckered. At first the lids of the left eye were much swollen; when the swelling had somewhat subsided the vision of that eye was found also very defective. When I examined him, three months after the injury, the left eyeball was sunken in the orbit, probably from cicatricial contraction of the orbital tissue. He had only the barest perception of light, and hardly any red reflex could be obtained from the fundus. I think he must have had extensive hæmorrhage into the vitreous chamber.

His condition, as you may easily imagine, was a most pitiable one. The only way he could be communicated with was by touching his fingers. And the only enjoyment he could have in life was through the sense of taste.

The next case was that of Corporal C—, who was on February 8th, 1900, at the battle of Paardeburg. He was crawling about on his stomach, after the manner of the soldier of the present day, when running short of ammunition, he raised himself to secure some more; at once he received a Mauser bullet through his head. It entered on the right side just above the zygomatic arch, midway between the ear and the outer margin of the orbit. It passed out on the left side through the frontal bone just above the external angular process. The man did not lose consciousness, but on being hit raised himself a little higher, and at once got another bullet through his shoulder. His lower jaw became fixed, and for seven weeks it had to be forced open to admit food. He said the sight of his left eye went within the course of about twenty-four hours. The lids of the right eye became much swollen, and he does not know how soon after the injury that eye went blind.

When I saw him on August 9th, 1900, six

Digitized by Google

months after the injury, there was partial ptosis of the right upper lid and defective movement of both eyes.

He had no perception of light with either eye. In the right eye the optic disc was grey in colour and the retinal vessels small; there was a large area of choroidal atrophy with retinal pigmentation to the outer side of the optic disc.

In the left eye an extensive white mass, probably decolourised blood-clot, obscured the optic disc and the larger part of the fundus from view; some patches of choroidal atrophy were visible. The choroidal atrophy was probably explained by injury to the posterior ciliary arteries.

The third case was that of a seaman, who, while in China with Admiral Seymour's relief expedition for Pekin, was hit on the head with a bullet. For a month he was unconscious, and had several attacks of bleeding from the nose and mouth. On regaining consciousness he found he was quite blind. There was a scar an inch and a half long on his right cheek, below the margin of the right orbit and running up and in to the side of the nose. On the left side of the head was a second scar, circular in shape, two inches above the zygomatic arch.

He had no perception of light in either eye. The left eyeball was displaced backwards in the orbit, and its movements in all directions restricted.

In the right eye the optic disc was greyish white in colour and the retinal arteries small; there was a rupture of the choroid below the disc. In the left eye the optic disc, as in the last case, was hidden by a white mass in the vitreous chamber; elsewhere the retina was detached.

It is, I think, interesting to note that the survival of these patients after these severe injuries is not due to the increased force or alteration in shape of modern bullets; for in Mackenzie's well-known book on diseases of the eye he quotes Dr. Thomson as having seen eight or ten very similar cases after the battle of Waterloo, and refers to two or three more after other engagements.

It is remarkable how very tolerant the orbital tissues sometimes are of foreign bodies which have become lodged in them. Shot which enter the orbit and are retained there rarely give rise to any trouble. Several cases of curious foreign bodies which have been found located in the orbit are on record. I showed, at the last consultation which I held here, a lad in whom a cartridge case was found embedded in the upper and inner part of the orbit.

I remember seeing my colleague, Mr. Tay, remove a piece of slate pencil nearly two inches long from the orbit of a boy. The history given was that six weeks previously he had tumbled downstairs, but nothing was said about any injury to the eye or any slate pencil. He had been treated for conjunctivitis. The presence, however, of what appeared to be a sinus at the outer canthus excited suspicion. On its being probed and dilated with forceps a rough substance was felt, and the piece of slate pencil drawn out.

At one of the first consultations which I held at the Polyclinic I showed a man who had had his common carotid tied for an arterio-venous aneurysm of the orbit.

Thirteen and a half years before I saw him, a revolver he was holding accidentally went off, the ball striking him just beneath the lobe of his left ear. The wound healed up, and he had no trouble until two years later, when the left eye gradually became prominent. For the past eleven years the amount of prominence had not increased, though it varied in degree from time to time. When he was actively moving, or excited, it became more marked, and there was bleeding from the left nostril. I have already mentioned how he was constantly troubled by the buzzing noise in the head.

This man, one Sunday, was reading his 'Lloyd's Newspaper,' when he saw it stated that an X-ray department had been established at the Moorfields Hospital. It occurred to him that by means of the X rays the bullet in his head might be discovered, and possibly some relief afforded him for the constant buzzing noise. He presented himself, therefore, at the hospital on the following morning.

On examination there was then found to be considerable fulness of both lids, especially the upper, and an irregular prominence extending to the side of the nose. The eyeball was much proptosed and its movements limited; the blood-vessels on its surface were enlarged.

On palpation the swelling was found to pulsate and present a distinct thrill. On pressure on the carotid the pulsation stopped and the swelling decreased in size. The optic disc was somewhat pale, and the retinal vessels small; V. of L. = $\frac{6}{24}$, of R. = $\frac{6}{6}$. The case was evidently one in which,

Digitized by Google

as the result of the injury, the carotid artery had ruptured into the cavernous sinus, so that blood from the carotid was being pumped into the veins of the orbit, causing them to become much dilated and to pulsate.

Dr. Mackenzie Davidson took a skiagram with the X rays for me, and localised the bullet as lying just below the basilar process of the occipital bone. I first tried the effect of continuous digital compression of the carotid by relays of assistants, keeping it up for six and a half hours, but with no permanent effect. Subsequently the man was transferred to the London Hospital, where Mr. Openshaw tied the left common carotid. For some time the effect was most satisfactory; all pulsation and the buzzing noise ceased, and the prominence of the eye was much decreased. Eight months later, however, a considerable amount of pulsation and proptosis had returned, but the noise was nothing like so troublesome as it had been before.

Other forms of vascular growth are met with in the orbit besides arterio-venous aneurysms. Some of these are congenital and of a nævoid character, and some, which are first met with in later life, are due probably to a sort of varicose condition of the orbital veins. I have met with two cases the symptoms of which are best explained by the assumption of such a condition.

In one, when the patient stood upright the eye on the affected side was unduly sunken in the orbit. In the other it was in the same position as its fellow eye. In both cases, when the patient stooped down, or when the jugular vein was compressed, the eye on the affected side gradually became proptosed. So that in the first case a condition of enophthalmos was converted into one of exophthalmos. In neither case was any pulsation to be felt or bruit to be heard. In the first case I think a dilatation of the veins must have caused some absorption of orbital fat, so that the eye became sunken; when, however, the veins were turgescent, then it became forced forwards. The second case was a charwoman æt. 51, and as her occupation involved a great deal of stooping in washing floors, etc., the coming forwards of her eye, which was always accompanied by a certain amount of pain, was a very troublesome symptom.

Cystic growths of the orbit are by no means common. Hydatid cysts are sometimes met with in this situation, and also dermoid cysts; these latter are, however, much more frequently located about the brow or in the eyelid. I had a case not very long ago in which there was what felt like a cystic growth at the upper and inner angle of the orbit. As, however, it was situated at the upper and inner part, and the eyeball was displaced down and out, I said, before operating, that very likely it would turn out to be a distended frontal sinus. When I cut into it the thick grumous fluid characteristic of frontal sinus secretion began to escape. What had happened was that the bony wall of the sinus gradually became thinned and ultimately perforated, so that some of its retained secretion escaped beneath the periosteum and formed the elastic cyst-like swelling. At the same time that this patient was in the hospital, I had another case presenting a hard mass in the orbit in the same situation, up and in. I treated him with iodide of potassium for about a fortnight, but the swelling did not show much tendency to decrease in size; then, from its position thinking it was probably a distended frontal sinus, I cut down on it. As soon as I had made an incision I found that what I had to deal with was evidently a diffuse new growth superficial to the bone, so I cut a piece of it out for microscopical examination. The sections of it showed the appearance of a diffuse gummatous infiltration. More rigorous treatment with iodide of potassium and mercury was instituted, and ultimately the swelling disappeared.

The above two cases show that often there is much difficulty in diagnosing from the symptoms alone the nature of an orbital growth. I may mention a third case to emphasise this point. It was one in which I saw a surgeon prepare to exenterate the orbit for what was diagnosed as an orbital sarcoma. The skin incision was commenced, but when the knife came to the upper and inner part of the orbit it slipped back into a distended frontal sinus just in time to save the eye from being enucleated.

Syphilitic periostitis is one of the commonest of all causes of swellings in the orbit. It is a late symptom of the disease, and may occur either in its acquired or inherited form. The margins of the orbit are the most prone to be affected, but it may occur in its deeper parts.

It is a good rule in all cases where orbital swellings of a doubtful nature are encountered, to give a thorough trial to treatment with iodide of

Digitized by Google

potassium before attempting any operative procedure.

An ivory exostosis in the orbit may sometimes be mistaken for periostitis or for a distended frontal sinus. The extreme slowness of its growth, and the markedly unyielding sensation to which it gives rise on pressure, are generally sufficient to distinguish it. In one case I had a skiagram taken with X rays, and the shadow of the bony growth was distinctly visible. I have never seen an ivory exostosis of the orbit upon which I have been tempted to operate. Those I have seen have had a broad base attached to the roof of the orbit. In such cases the growth often begins in the frontal sinus, and extends not only downwards into the orbit, but also upwards into the cranial cavity.

Before concluding this somewhat miscellaneous collection of clinical notes, I must refer to the operation of exenteration of the orbit—an operation which has to be resorted to in cases of malignant disease. It is not, however, in every case of malignant disease of the orbit in which the whole of its contents has to be taken away and the eyeball sacrificed. I have already mentioned one case of sarcoma of the orbit where the growth was encapsuled, and where by means of Krönlein's operation I was able to get it all away and retain a useful eye. In some early cases of malignant disease of the lachrymal gland it is possible to remove all the growth without taking out the whole contents of the orbit. It has also been shown that by Krönlein's operation a tumour of the optic nerve can be removed and the eyeball retained, though, of course, sightless.

Cases in which I have myself performed exenteration of the orbit are those where there has been a deeply seated, very extensive, or diffuse sarcoma; or where there was a rodent ulcer involving so much of the eyelids that its removal was impossible without leaving the cornea exposed and liable to ulcerate.

In exenteration of the orbit it is best to remove the new growth, the eyeball and its muscles, together with the conjunctival sac, and margins of the eyelids all *en masse*.

In sarcoma of the orbit it is possible to retain the eyelids, but though possible, not desirable. After the operation no artificial eye can be worn; the upper lid is paralysed and hangs as a useless pendulous flap, which in the subsequent treatment is always in the way. The conjunctival surface is, moreover, the perpetual source of an annoying and troublesome secretion.

If the margins of the lids and the conjunctival sac are removed, the whole cavity of the orbit cicatrises over, the contraction on the neighbouring parts causing the eyebrow to be drawn down and the soft tissues of the cheek upward, so that it is surprising how little deformity is ultimately left. The procedure of the operation is as follows:—The patient having been anæsthetised, an incision is made through the skin of the eyelids, about ten millimetres from their margins, around the whole palpebral aperture. If the new growth involves the skin of the lids, the incision has to be diverted so as to pass through healthy skin well beyond the affected area. Sometimes it may be necessary to make it extend down into the cheek or on to the bridge of the nose.

The incision having been made, the skin is next dissected up as far as the margin of the orbit in all directions. The margin of the orbit having been reached, the palpebral ligament attached to it is cut through in its entire circumference close to the bone. A stout pair of scissors, curved on the flat, is then taken and, directed by the finger, passed backward along the outer side of the orbit, close to the bone, to its apex. A few rapid snips are then made to divide the structures at the apex; the eyeball, conjunctival sac, margins of the lids, growth, and other orbital contents being at the same time drawn forwards in one mass. Any structures attaching it to the walls of the orbit are to be divided close to the bone until they are entirely separated.

It is remarkable with what facility the orbital contents down to the bone can be cleared out in this way. The hæmorrhage from the back of the orbit is always free, but is easily controlled by pressure.

Queen Anne Street,
February 1st, 1904.

Hyoscin Hydrobromate.—Abramson ('New Orleans Med. and Surg. Journ.,' December, 1903) advocates the use of hyoscin hydrobromate injections in cases of morphinism. He believes in the use of tonics, etc., for some time after the morphin has been finally withdrawn and the effect of hyoscin has passed away.—*Journ. A. M. A.*, January 2nd.

Digitized by Google

A LECTURE
ON
SOME POINTS IN BALNEARY THERAPEUTICS.

Delivered at the Medical Graduates' College,
By LEONARD WILLIAMS, M.D., M.R.C.P.,
Assistant Physician to the German Hospital; Editor of the 'Journal of Balneology.'

GENTLEMEN,—In two lectures which I have already given at this College* I dealt with some points in climatology, and when I was invited to give this one, I thought to supplement what I then said by further references to the same subject. On consideration, however, I have deemed it better to leave the amplification of that matter to another occasion, and to ask your attention to-day mainly to the principles which underlie the allied science of balneology.

Climatology and balneology belong to a triumvirate, the third member of which has, curiously enough, never been christened. This is the science of prescribing natural mineral waters for internal administration, the science, in fact, of spa treatment, properly so-called. In a recent American work the rather uncouth name of "Crounotherapy" has been suggested (from χρουνος, a well, and θεραπεια, healing). A better word, I am informed, would be Crenology. It is shorter and more euphonious, and as a word is badly wanted, I shall employ it. Now, it is important to remember that these three, climatology, balneology, and crenology, are to a large extent interdependent. Climatology may be made to stand alone, no doubt; but neither balneology nor crenology can be considered apart from climatology. A change to a suitable climate may fulfil all the indications presented by a particular case, but if we wish to subject our patient to treatment by baths and waters, it is necessary for us to consider carefully whether the climate of any place which suggests itself will reinforce the treatment by baths and waters, or whether it will have the contrary effect; whether, in fact, the climatic modifications, if any, will further or retard the treatment of the particular case. Neither can treatment by baths and waters, respectively, be really dissociated, for, in the hands of the skilful physician, the one can nearly always be so manipulated as to heighten the beneficent effects of the other.

Now, the domain of pathology, over which this triumvirate may be said to hold therapeutic sway, is roughly comprised in the term defective metabolism. There are other regions, of course, in which the discipline is conspicuously beneficial, such as syphilis and some forms of heart disease. With the latter I shall deal presently, but I would say here with regard to syphilis that the Aix-la-Chapelle method of treating it by inunction, baths, and an open-air life, is as much superior to the ordinary drug treatment as the open-air treatment of phthisis is superior to that by the hypophosphites; and I would even go so far as to say that a man is not doing his duty by a patient with recent syphilis unless he urges him to go through a course of treatment on the Aix-la-Chapelle model. This can be obtained in London; but it is much better to send such cases to a health resort, and you may have every confidence in its being properly carried out at Harrogate, Bath, or Sidmouth.

But, speaking generally, the signs of defective or faulty metabolism are the signs by which you may recognise the patient who is most successfully treated by climates, baths, and waters. Now, what these signs are I need not stop to inquire. Under the titles gout, goutiness, rheumatism, arthritis, and so on you are as well acquainted with them as I am. Clinically they are sufficiently easy of recognition, but they are nevertheless often very difficult to label. They are due for the most part to a want of balance between intake and output, and for cases occurring amongst the sedentary dwellers in large cities by far the most efficient means of redressing that balance is the triumvirate of which I have been speaking. To climatology and some of its possibilities I have already referred; with crenology I may perhaps deal on a future occasion; to-day I must content myself with some brief references to the principles which underlie the practice of balneology.

To the question, "What is balneology?" I would reply, balneology is the recognition and utilisation for therapeutic purposes of the functions of the skin. Now, the skin is by far the largest organ in

* Published in the CLINICAL JOURNAL, August 26th and September 16th, 1903.

Digitized by Google

the body, and its functions are both numerous and complex; and yet it is, of all the organs of the body, the one which has suffered, and still does suffer, the most systematic neglect at the hands both of the public and the profession. How many people, for example, are careful about the excretory function? The liver they will cultivate with the most vigorous assiduity, stimulating this astonished viscus with every new drug and nostrum which commercial energy places on the market; the kidneys they will discuss, and their secretions investigate with all the explorer's zeal and enthusiasm; but the only attention vouchsafed the skin is that of woollen garments, chest-protectors, and cosmetics. The excretory power of the lungs is little enough regarded, in all conscience, especially when we consider the equanimity with which persons who are otherwise sane, sober, and cleanly will continue to breathe and rebreathe an atmosphere which has passed through the ultimate air-cells of other people. But, thanks to the open-air treatment of phthisis, the sinister importance of the pulmonary excretions are rapidly gaining recognition, whereas the eliminative function of the skin still languishes in the cold shades of habitual neglect. I say "habitual" neglect, because this neglect is not complete. In some conditions, advanced cirrhotic kidneys for example, its aid is almost invariably invoked, and the assistance which it then gives in ridding the system of the uræmic poison might well point the moral of its every-day importance.

Of what, exactly, the cutaneous secretion consists we do not really know. The analysable constituents are given in the text-books on physiology, and from a study of these it is evident that the really important ones have so far escaped recognition. In these same text-books, however, an experiment is described which is full of instruction on this point. We all remember the animal the whole of whose skin was varnished and who succumbed, in consequence, after a few days, with great depression of temperature. Sir Michael Foster regards these symptoms as due to toxæmia, and from a clinical standpoint there can be no doubt that this is the correct view. It is therefore evident that it is the business of the skin to eliminate some principle whose retention is deleterious, and ultimately fatal, to the economy. Now, how many of us realise this fact? We may accord it a sort of perfunctory academic recognition, but very few amongst us treat either ourselves or our patients as if the excretory function of the skin were of any real importance. We point to the sedentary lives of town-dwellers as accounting for all metabolic troubles, but we always lay the blame on the stomach, liver, or bowels. Balneologists attribute to the skin its full share of responsibility in these matters, and enlist its powerful aid in restoring the normal balance. That is one of the reasons why health-resort treatment is so successful, especially as compared to measures in which the excretory power of the skin fails to receive its due share of attention.

There are two other functions of the skin to which I must briefly refer. The one is its heat-regulating power, the other is its power, through the nerve-endings, of passing on a stimulus to surrounding tissues or to distant organs. A familiar illustration of this last is the action of a blister. Although no one has yet offered a satisfactory explanation of how a blister acts, no one has been found to contest its power of modifying the trophic processes in its immediate neighbourhood. The same may be said of liniments and other stimulating applications. They act by affecting the nerve-terminations, which, in their turn, exercise an influence upon the neighbouring tissues. This we know, but of the exact *modus operandi* we are ignorant. Now, it is obvious that balneary processes generally, in that they stimulate these nerve-endings, must all of them exercise an effect similar in kind, though less in degree, to the effect produced by blisters, liniments, and the like. The influence which general baths (that is the immersion of the whole body) may have upon internal organs has been dimly appreciated since the earliest times. It used to be thought that its influence was due entirely to the alteration of the main blood-stream from centre to periphery, from internal organs to skin, and *vice versâ*. This, no doubt, is a very powerful factor in the situation, but the part played by the terminal nerves in producing reflex stimulation of distant organs has been brought home and emphasized by the success of the Nauheim treatment of heart disease. Too much has certainly been claimed for this treatment, but the power of the baths to reduce chronic dilatation is beyond all question. Neither can there be any question as to the effects upon other organs and structures of the stimulation of these

Digitized by Google

nerve-endings by other balneary processes. How else are we to explain the remedial effects of these processes in those chronic joint conditions, whether they be due to gout, rheumatoid arthritis, or chronic rheumatism, in which balneology achieves by common consent its most striking and lasting results? These effects used to be attributed to the local application of heat, but we now know that heat is but one among the many agencies which we possess to this end, and that there are others which are almost equally potent in the direction of producing this stimulation of the nerve-endings, and consequently of modifying the trophic processes. Consider for a moment what these means are. First, there is the temperature of the water, and in this connection it must not be forgotten that cold is an even greater stimulant than heat; secondly, its composition as to constituent salts and gases; thirdly, its method of application, whether by reclining bath, douches, showers and needles, either alone or combined with massage; and finally, the accessory means, such as hot air, light in various forms, and electricity, with which all modern bathing establishments are fully equipped. We, who have practised these methods, know their effects, and can afford to smile at the scepticism of the sceptics. To explain the why and wherefore of these effects is, however, at present impossible. But that is merely a question of time.

With regard to the heat-regulating function of the skin, I have only this to say—that if it is to do its work properly it must be kept in proper training. The majority of elderly people and many young ones, so swathe themselves in flannels and furs during the cold weather as to prevent any cold influence ever reaching the cuticle. In this way the skin either loses altogether its power of contraction or contracts so sluggishly to a stimulus that when the cold influence comes, as, some day, come it must, the loss of heat is so considerable as to cause serious trouble. Properly directed balneary treatment is a perfectly safe and most efficient means of counteracting the pernicious effects of the too common tendency to coddle. It hardens the hot-house plant and enables it to live in the open. If people would only educate their skins to do this work properly and place some confidence in the contractile power, there would be less of that fear of draughts which renders the majority of rooms so intolerably stuffy.

In this connection I presume I am expected to say something about the absorptive power of the skin. Well, so far as the absorption of the ingredients of natural mineral waters used for bathing is concerned, this power does not exist; or, if that is overstating the case, it does so in a degree so slight as to be altogether negligible. That the skin possesses this power we need go no further than the inunction treatment by mercury to assure ourselves; but it is practically certain that, whatever the extent of that power in other directions, it fails to include the absorption of the ingredients of natural mineral waters in any material degree. After all, if this absorption is desired, it is so much simpler to give these waters by the mouth.

Now, the application of these principles and methods to the individual case is necessarily a matter for experts, and the exact balneary procedures most likely to benefit a particular patient are best left to the physician to whom the case is sent at the selected spa. And this leads me to emphasise the necessity of writing to the physician, in order to acquaint him as far as possible with the history and general circumstances of the case, and of setting forth the facts on which you have based your decision to recommend spa treatment. Neglect of this simple precaution often leads to differences between the physician who has recommended the spa and the one whose duty it becomes to carry out the treatment.

For, if you wish to obtain the full benefit of a balneological course you must send your patient away. In the first place, the proper means for such treatment are not to be had in large towns; and in the second, such treatment is liable to be unsuccessful unless the patient relinquishes his ordinary life and gives himself up to follow out its details. These details include regular hours, dietetic regulations, and often the assistance of crenological processes. It is the experience of every one who has practised at health resorts that even patients who are most *difficile* at home become lamb-like in their docility when subjected to a course of treatment which is strange to them, and which the majority of their fellow-visitors are undergoing. It is only the *habitué* of a particular spa who knows more about his ailments and the effects upon them of the waters than the doctor.

Digitized by Google

If his medical adviser is wise, he will find cause to vary the spa of such a person from time to time.

There are nevertheless some balneary procedures to which I may direct your attention as suitable for the purposes of practice in places where there is no regular bathing establishment. The first of these is the Turkish bath. As most people are familiar with it, this is a process I need hardly describe to you. It is a dry hot-air bath followed by massage of a crude sort, and terminating with ablutionary measures and a tepid or cold douche or spray, or a plunge into a swimming bath. The objection to these baths as given in institutions is that the ventilation is invariably insufficient, so that if there are many other bathers in the hot rooms the odour of cutaneous exhalation is unpleasantly apparent. There are many "home Turkish baths" now on the market, and although these have drawbacks as compared to institutions, they have the great merit of enabling the bather to breathe pure air. Their drawbacks are that they lack the elements of shampooing and douching which, in a well-administered establishment, constitute such important and agreeable features of the bath. If the bath is taken at home, the sweating process should always be followed by abundant soaping and by the application, in an ordinary bath, of water at about 60° F. A Turkish bath once a week is an admirable means of promoting metabolism in an otherwise healthy person whose opportunities for exercise are unduly small. But it is only for the comparatively healthy that they are suitable. No one with disease of the heart or arterial system, except perhaps slight atheroma, should be allowed to take them. In commencing granular kidney, before the heart has begun seriously to feel the strain, they constitute an excellent routine measure.

Another balneary method which can now be obtained in most large towns is the Nauheim treatment. As everyone now knows, this consists of tepid, gaseous, saline baths, with resisted exercises. There are many institutions and nursing homes in and about London to which patients may be recommended with every confidence that the treatment will be administered by properly trained and experienced attendants, but I should neglect my duty if I were to fail to impress upon you the very great advantages to be obtained by sending such patients to the quiet, the routine, and the discipline of a properly equipped health resort. There is no necessity to send these cases to Nauheim. The treatment is admirably carried out at Harrogate, Buxton, Matlock, Strathpeffer, Llandrindod, and Llangammarch, all of which are bracing places; and at Bath and Sidmouth, which are sedative stations. It is never well to send such patients across the sea if this can be avoided.

Now, with regard to the indications for these baths I must permit myself a few words. They have been extolled in all diseases of the circulatory system, but their application is in reality limited to chronic dilatation of the heart, whether or not this be accompanied by valvular disease. And first of all let me insist upon the word chronic. Any acute or subacute element constitutes an absolute counter-indication. I know that this is not the opinion of some, but it is very emphatically my own, and I strongly recommend you to adopt it as yours. When the disease is recent or when, at the opposite end of the scale, compensation is altogether lost, these baths do harm. Bed, digitalis, leeches, and the like are the only proper and safe means to adopt in such cases. Aneurysm and extensive arterio-sclerosis are also absolute counter-indications. In dilatation without valvular lesion or in commencing loss of compensation you may, on the other hand prescribe these baths and their concomitant exercises with the utmost confidence in their power to effect a real and comparatively lasting improvement. These are conditions which are, however, very frequently overlooked precisely at the stage in which the baths would be productive of most good. Nine men out of ten, when percussing the heart's area, satisfy themselves with ascertaining the amount of superficial dulness. Now, I ask any thinking person, what information is to be obtained by finding out the amount of the cardiac muscle which is uncovered by lung? For that is all that superficial dulness tells you. What you want to know is the exact size of the heart, and this you can make out easily enough, with a little practice, if you will use a light percussion stroke, the lightest, in fact, which will elicit a note at all. The sledge-hammer style of percussion may have merits, though I have never been able to discover them, but for mapping out the size of the heart it is altogether valueless. With a light stroke, the normal area of dulness, which corresponds with the actual size of the heart, begins on the left side, in the third interspace, close to the

Digitized by Google

sternal margin. The normal position of the apex-beat should correspond with the dulness in the fifth interspace. On the right side the normal dulness begins in the fourth interspace, about the length of the fore-finger nail (1½ centimeters) to the right of the sternal margin. It is at this point that slight dilatation most often shows itself, and if you fail to practice the only sure means of ascertaining such slight dilatation, namely, light percussion, you will most certainly miss the indications for the most favourable cases for treatment by the Nauheim method.

The next procedure to which I would ask your attention is the hot wet-pack, than which there are few better methods for promoting metabolism through the agency of the skin in the very young, the aged, and the weakly. It is an old method which has become unduly neglected. It is applicable in any condition where metabolism is defective or where it is desired to relieve the system of retained poison. In anæmia I know of nothing better; in the sequelæ of scarlet and other fevers I know of nothing better, and in slight catarrhs of the air-passages in children it has no superior. For convulsions in children it is infinitely neater and more agreeable than the ordinary hot bath. The child is kept quiet, and its head is more easily kept cool. All that is required is a mackintosh sheet, two ordinary blankets, a cotton sheet, a hot-water bottle, a pail of boiling water, and a wringer; all of which, except the last-named, can be obtained in an ordinary household. The wringer can be manufactured at short notice by hemming in enough of the two ends of a towel to allow of a walking-stick to be passed through each end easily. The mackintosh is placed on the bed and on top of it the two blankets fully spread out. The sheet having been rung out of the boiling water, is then laid on the upper blanket. The patient is then placed on the sheet so that his occipital prominence is on the upper margin of the sheet. With the patient's arms raised, the upper corner of the sheet on his right side is carried across and tucked under his left scapula. With his arms by his side the opposite corner is brought across and tucked under his right scapula. Each blanket is then carried across in a similar manner, the hot-water bottle is placed near the feet, and the free ends of all the coverings are tucked under the heels. In a period varying from twenty to forty minutes perspiration will be found on the forehead, and that is the signal of sufficiency. The temperature taken in the mouth generally shows a rise of one or two degrees. After a tepid or cold sponging the patient is removed to bed, on which the coverings should not be too heavy. Two or even three of these packs may be given in a week. Patients, especially children, nearly always like them, and an intelligent nursery nurse can be taught to administer them quite satisfactorily. They are infinitely superior to ordinary hot baths, and are not nearly so exhausting.

The last thing which I have to bring to your notice is the Dowsing radiant heat bath. In this bath the skin is subjected to the influence of light as well as heat, and it is wonderful what the addition of light seems to confer upon the action of the heat. In a bath of this kind, when the thermometer registers 300° F. or even more, one is not conscious of anything more than an agreeable warmth, and yet the skin acts quickly and profusely. I know of no method of promoting active metabolism with so little demand upon the patient as this bath. It is very useful in obesity, in chronic metallic and other poisoning, and in chlorosis; but it finds perhaps its most striking success in its power of bringing about the absorption of gouty and rheumatic deposits. It is only fair to the Dowsing Company to point out that this is not an ordinary incandescent electric-light bath. Their lamp is a patent for which it is claimed that it not only gives out infinitely more heat than the ordinary lamp, but that the rays have far greater penetrating power. These claims the spectrum would certainly seem to support. This bath, when followed by efficient massage and the soaping and douching which should attend all sweating processes, is much enjoyed by patients. It has, however, the disadvantage of a rather limited applicability, in that it is by no means inexpensive.

York Street, Portman Square, W.
January 29th, 1904.

Supra-renal Extract.—Migliacci ('Gaz. deg. Ospedali. Milasa, No. 134) presents arguments to prove that supra-renal extract is as much a specific for akinesis of the muscular system as strychnin is for akinesis of the nervous system.—*Journ. A. M. A.*, January 2nd.

Digitized by Google

A DEMONSTRATION OF CASES

AT THE

MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

By BYROM BRAMWELL, M.D., F.R.C.P.E.

(*Concluded from page* 240.)

CASE 3. *Lesion involving (?) the sympathetic and recurrent laryngeal nerve or vagus on one side; ? syphilitic.*—The next patient, a married woman æt. 40, was brought to the clinique by Dr. Dundas Grant, who described the case. The patient had suffered from the symptoms since marriage. She has had "spasms in the throat" for three years, and is now short of breath. She complains of choking in her throat. There is a very marked difference in the size of the pupils; the right pupil is much larger than the left. The left side of the face perspires more than the right side. The left palpebral fissure is narrower than the right, and there is some compensatory elevation of the left eyebrow and wrinkling of the left side of the forehead. There is no loss of power in the orbicularis palpebrarum. The uvula is a little swollen. When the patient phonates, the uvula seems to be drawn slightly to the right side. Phonation is markedly affected. There is very incomplete movement of the vocal cords, especially the left one—it is flaccid. The patient has had seven children; they are all healthy. There have been no miscarriages. She improved while she was taking iodide of potassium. She, however, left it off, and the symptoms have returned to some extent. The condition of the larynx is suggestive of an affection of the recurrent laryngeal nerve or of the vagus higher up. There is no evidence of intra-thoracic pressure. There is no pain in the chest, or cough. There is, Dr. Grant says, no evidence of a thoracic aneurysm. There is no tumour or swelling in the neck. There is no evidence of intra-cranial lesion; there has been no headache and no vomiting; but she is giddy. The optic discs are normal. Her hearing is quite good. She says she feels nothing wrong with the arms or legs, except stiffness. She says she is not very steady on her legs, but she states that she has no difficulty in walking. Her knee-jerks are present, but not very active. It is a very interesting case, but the diagnosis is difficult; perhaps there is a double lesion. The "style of the symptoms," the facts that the condition developed after marriage and that the symptoms improved under iodide of potassium, seem to suggest a "specific" lesion.

CASE 4. *Hemianæsthesia with peculiar changes in the fields of vision.*—The next patient was also kindly brought by Dr. Dundas Grant. The patient is unmarried, a schoolmistress, æt. 42 years. She has been ill altogether eleven years; she has suffered from suppuration in the sphenoidal sinuses, which have been operated upon; she has also been under treatment from time to time for her voice; she has had several attacks of functional aphonia. She now complains of headache, and there is right-sided hemianæsthesia. The hemianæsthesia was first detected last July; it may, of course, have been present before that date, but if so the patient was not aware of it; it was accidentally discovered. It has been constant since. There is complete loss of sensation to touch, pain, and heat and cold on the right side of the body—the face, arm, and leg. The loss of sensation to heat is so marked that the patient states she has frequently burnt her hand without feeling the burn. That seems to me peculiar and exceptional. I do not remember to have seen any case of functional (hysterical) hemianæsthesia in which the patient has burnt herself without knowing it. There is complete loss of the stereognostic sense on the affected sides. A variety of articles (pencil, knife, buttonhook, tuning-fork, shilling, etc.) were in succession placed in her right hand, and she was unable to recognise and distinguish them by touch when the eyes were closed. In a case like this, in which all the primary sensations are lost—pain, touch, heat and cold, etc.,—one would naturally expect that the stereognostic sense would be abolished. Whether the muscular sense is abolished I do not know; the patient's power of appreciating the difference between different weights, etc., should be tested. She feels heat and cold quite well with the other (left) hand. On testing the sensibility to pain it was noticed that the area of loss of sensation on the face was not sharply limited to the middle line; it extended for a considerable distance (a couple of inches or so) to the left of the middle line on the forehead and upper lip. Taste, hearing, and sight were defective

Digitized by Google

on the right side. (Owing to the local disease in the nose, smell could not be tested.) The fields of vision were then roughly mapped out on the blackboard. In the right eye the fields for white, red, and blue were markedly contracted. In the left eye the constriction for white was still more marked than in the right eye, and red and blue were not perceived by the left eye (yellow and green were not tested). The condition of vision is quite peculiar and remarkable; the fields are notably constricted on both sides, but the constriction is not, as one expects to find in a case of functional hemianæsthesia, most marked on the side of the anæsthesia. In functional (hysterical) hemianæsthesia there is often amblyopia with constriction of the fields for white and for colours in both eyes, but the constriction is most marked on the side of the hemianæsthesia. In this case the constriction is most marked on the left side (the non-anæsthetic); the patient cannot perceive red and blue with the left eye. That is a condition I am not acquainted with in hysteria, and I am not aware that any case of the kind has been described. There has been no vomiting; there is no optic neuritis; but the patient is a good deal troubled with giddiness. She says that she is not emotional, but that she is "neuralgic." There do not appear to be any signs of any organic lesion of the nervous system. The general health is fairly good. The patient suffers from headache, but that, Dr. Grant thinks, is accounted for by the condition of the frontal sinuses. The knee-jerks are very well marked, but equal on the two sides. The plantar reflex shows no toe movement on the right (hemianæsthetic) side, but flexion on the left (sound) side. There is a little uncertainty in performing the finger-nose test.

Let me sum up the facts of the case so far as I know them. The patient, a single woman æt. 42, has for several (eight) years suffered from suppuration of the frontal sinuses; she has had several attacks of temporary (functional) aphonia; she suffers from headache and giddiness; she is, she says, not emotional. Some three months ago it was discovered that the right side of the body was anæsthetic. The hemianæsthesia has been constant since that time; it is very profound, and affects all forms of sensation (touch, pain, heat and cold, the stereognostic sense). The loss of sensation to pain is not sharply limited to the middle line, but extends on the face considerably farther over on the sound (left) side than is the case in most cases of hemianæsthesia. (It would be important to have sensation of all forms carefully mapped out and charted all over the body.) The loss of sensation to pain and heat is so profound that the patient has on several occasions burnt the right hand without knowing it and feeling it. This in a case of functional hemianæsthesia is, in my experience, peculiar. Sight, hearing, and taste are impaired on the anæsthetic side. The fields of vision are very peculiar, and quite different from anything I have seen or heard of in functional hemianæsthesia. The fields are constricted in both eyes, but the constriction is most marked on the non-anæsthetic side, and on this (the non-anæsthetic) side the patient cannot recognise red and blue (*i. e.* with the left eye).

The great question, of course, is, are these symptoms functional, or are they due to organic disease? That is a question which is constantly cropping up, and it is a most difficult one in some cases to answer. I have not time to go into the case and consider the diagnosis in anything like detail. The hemianæsthesia is, of course, highly suggestive of a mere functional disturbance, a so-called hysterical condition; but if functional, the case is a very anomalous one because of the peculiarity in the fields of vision. It is peculiar and unusual. The absence of any definite signs of organic (intra-cranial) disease, the condition of the plantar reflex, and the history of functional aphonia no doubt point to a functional (hysterical) condition. If the hemianæsthesia were due to an organic lesion, one would of course expect that lesion to be situated in the neighbourhood of the internal capsule, to involve the posterior part of the internal capsule. But an organic lesion in this situation would in all probability be attended with some loss of motor power, especially in the leg, and with some alteration in the reflexes; the knee-jerk on the left (hemianæsthetic) side would probably be increased, and the Babinski sign would probably be present on the right (hemianæsthetic) side. Here there is an absence of toe movement; that is what one would expect in a case of functional (hysterical) hemianæsthesia. There is no evidence of a lesion involving the motor tract. There is no evidence of an intra-cranial tumour. Then a lesion involving the posterior part of the

Digitized by Google

internal capsule would not produce the alteration in the fields of vision which is present in this case; it would produce homonymous hemianopsia.

Another thing which suggests itself as a possibility is syringomyelia. In that disease I have seen the loss of sensation chiefly limited to the face, arm, and leg on one side, but I do not know of any case in which the sensory loss was, practically speaking, entirely hemiplegic as it is in this case. Again, the loss of sensation in this case involves all forms of sensation—touch, as well as pain and heat and cold,—and is absolute on one side of the body. That is not like syringomyelia. In syringomyelia one would not expect such a profound disturbance of the sense of touch all down one side of the body. Further, in syringomyelia the special senses (sight, hearing, taste) on the side of the anæsthesia are not affected as they are in this case. And again, in this case there are none of the other signs of syringomyelia; there is no atrophy of the small muscles of the hand or of the shoulder-girdle; the knee-jerk is not increased, and there is no extensor response (conditions which are present in some cases of syringomyelia); and the patient does not present any of the alterations in conformation and physique which one so often finds in syringomyelia. My feeling is that the case is functional, but that it is a very unusual form of functional disease. I do not pretend, after a hasty examination of this sort, to get at the bottom of such a case. I would suggest that the sensory fields and the fields of vision should be very carefully mapped out. A long time would be required to make an accurate examination of that sort.

Shock.—According to Kinnaman's experiments and studies, shock must not be considered as due to a lowering or exhaustion of one bodily function, but the composite result of interference with the blood-pressure and respiration, lowering it in both cases, and a marked fall in the body temperature. As it increases in severity the fall in temperature is the most uniform and progressive symptom. The relationship between the lowered body temperature and shock is evidenced by considering—(1) that in one of the series of cases he reports the fall in temperature was the sole cause of the shock; (2) that where by continuous bath the temperature fell only 1° C., the respirations were increased instead of diminished, and the fall in the blood-pressure was lessened; (3) that by raising the body temperature previously lowered in shock, the respiratory rate was increased and the blood-pressure raised. The relationship he expresses thus: (*a*) a sufficient fall in body temperature can cause a decrease in the respiratory rate and a marked fall in blood-pressure, which, together with itself, we designate as shock; (*b*) conversely, a limiting of the fall limits the fall in pressure and prevents a fall in the respiratory rate, thus limiting or preventing shock; (*c*) antagonistically, a rise of temperature causes a rise of blood-pressure and the respiratory rate (reduced in shock), thus ameliorating all the symptoms. Thus of the three factors temperature takes the first place by its power of production, its power of limitation, and power of amelioration of the composite condition—shock. —*Journ. A. M. A.*, January 16th, 1904.

Oppenheim's Reflex.—Oppenheim calls attention to a phenomenon which in significance is very close to the Babinski phenomenon, and which he himself first observed and described. In a normal individual, on stroking the inner surface of the leg from above downwards with either the thumb or the handle of a percussion hammer, or pinching a fold of skin on the inner surface of the leg, either no reflex movement will follow or a flexion of the toes will result. Under pathologic conditions—that is, in pyramidal involvement,—instead of the plantar flexion of the toes, dorsal flexion is observed. This reflex differs from the Babinski not alone in the source of its inception, but in the fact that not only the extensor hallucis longus participates in the reflex movement, but also the tibialis anticus and often the extensor digitorum communis and the peronei. The contraction of the extensors for the foot is physiologic when examining the plantar reflex, and the pathologic reflex is only known through the contraction of the extensors for the toes, whereas in Oppenheim's reflex the pathologic reflex is more plainly indicated by the involvement of other muscles. In a few cases where the estimation of the toe reflex is uncertain the Oppenheim reflex may be decisive.—*Medical Record*, January 2nd, 1904; *Journ. A. M. A.*, January 16th, 1904.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 589. WEDNESDAY, FEBRUARY 10, 1904. Vol. XXIII. No. 17.

CONTENTS.

PAGE

*On Common Diseases of the Liver. By W. HALE WHITE, M.D., F.R.C.P. ... 257

*With Dr. Campbell Thomson at Middlesex Hospital — The Trophic Changes in Locomotor Ataxy ... 264

*Appendicectomy. A DISCUSSION BY H. L. BARNARD, G. L. CHEATLE, JAMES BERRY, J. SHERREN, W. H. B. BROOK, H. RIGBY, A. MICHELS, W. H. BEST, and C. WALLACE 267

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London E.C.*

ON COMMON DISEASES OF THE LIVER.*

By W. HALE WHITE, M.D., F.R.C.P.

GENTLEMEN,—When your secretary kindly suggested to me the subject of common diseases of the liver I thought the simplest way to find out what were the common diseases of the liver would be to look through my case-books, and I learn that among my last two thousand patients the common diseases of the liver have been carcinoma, cirrhosis, and gall-stones, all of which have been frequent, about 4 per cent. of the two thousand cases being one of these three. The next most common diseases of the liver among my cases are the nutmeg liver of heart disease, and other forms (than gall-stones) of obstruction of the duct. Then, rarer, come syphilis of the liver, abscess of the liver, and perihepatitis; and lastly, there have been two cases of the large liver associated with malaria, and one case of acute yellow atrophy and one of hydatid. Now you will notice that there is no case of catarrhal jaundice, but that, I think, is because the sufferers from it are rarely sufficiently ill for it to be necessary to have a second opinion; it is not a very common disease in the wards of a hospital, probably because it is not severe enough for the patient to come into a hospital. The fact that no case of lardaceous disease was seen is to be attributed to the improved modern diagnosis of syphilis, and the correct treatment of it and long-standing suppuration. There is no case of fatty liver, but that is probably owing to the difficulty of detecting it during life.

Now although catarrhal *jaundice* is hardly seen in consulting practice, it is, I think, fairly common for patients suffering from it to come to the out-patient department of the hospital, and some are

* An address delivered before the Wandsworth Division of the British Medical Association.

Digitized by Google

occasionally admitted. You will, I think, agree with me that as a rule the diagnosis is not difficult. The age and general aspect of the patient usually prevent our calling the case one of malignant disease, and if this mistake should be made the gradual diminution of the jaundice will soon set us right; nor is cirrhosis often confounded with catarrhal jaundice. As a matter of practice, probably confusion most often arises between gall-stones and catarrhal jaundice; but gall-stones are common in elderly women, catarrhal jaundice is most common in young men. The pain of catarrhal jaundice is a dull and continuous discomfort, whilst that of gall-stones is paroxysmal and sharp, and the sufferer from this disease either has a short sharp attack of pain and jaundice, or a recurrence of attacks; while in catarrhal jaundice the trouble is for a time continuous, but gradually diminishing. The text-books constantly tell us that catarrhal jaundice is due to gastro-duodenal catarrh, the process spreading up the duct; this probably is so, but when authors also describe that catarrhal jaundice is nearly always preceded by symptoms pointing to gastro-intestinal catarrh, I cannot help thinking they are wrong. Catarrhal jaundice frequently appears without any immediately previous symptoms of gastro-intestinal catarrh. The liver is often somewhat tender and a little enlarged, owing to the damming back of the bile. The depression of spirits which is so often the result of the circulation of bile in the blood seems to be very pronounced in catarrhal jaundice. We must not be put off our guard by slight pyrexia, for that may certainly occur with catarrhal jaundice. There is usually a great loathing of food, and the patient in the early days of the attack is probably best on slop diet, and very little of it; he feels so seedy that he naturally keeps to his bed, and I believe he derives great benefit from a large dose of calomel at night, followed by a good saline purge in the morning.

A nutmeg liver is, in my experience, much more common in hospital than private practice. This is no doubt because hospital patients with disease of the heart have to work hard, and hence compensation soon fails. In passing it is worthy of note that a nutmeg liver is rarely seen as a result of pulmonary disease. The size that the organ attains in some cases of failing cardiac compensation is very great, and often a large nutmeg liver is the only, or almost the only, evidence of the over-filling of the venous system; but this is hardly surprising, for as soon as any of the veins begin to give way under the increased pressure they become the weakest part of the venous system, and hence continue to give way. As is well known, the liver not infrequently pulsates when the tricuspid orifice is incompetent, but it is surprising how often it is said to pulsate when really it is only heaved up and down by the enlarged heart or by the aorta. The only way to demonstrate that the liver pulsates is to feel a distinct expansion of it, one hand being placed in front of it, the other behind it. The other day I was assured that a liver pulsated in a case of aortic disease, but this was not so; it was simply thrown backwards and forwards by the big heart at each cardiac contraction. We are inclined to forget that only the diaphragm intervenes between the heart and the liver. I have now under my care at the hospital a case of aortic disease in which pulsation can be felt in the digital vessels of the terminal phalanges, but no pulsation can be felt in the liver, and as far as I know pulsation is never transmitted to the hepatic artery so that we can feel the liver pulsate. A nutmeg liver is often extremely painful; if this is so the pain may be at once relieved by putting on a few leeches over it and by giving a good dose of calomel.

A problem with which we are often confronted is that we have before us a patient past middle life who for some time has had jaundice. The cause of it is not apparent. Usually it is due to one of three causes: cirrhosis of the liver, malignant disease of the liver, or obstruction of the common bile-duct, and the likely causes of this are an impacted gall-stone or chronic pancreatitis. Of these, cirrhosis of the liver is undoubtedly the easiest to exclude. The history of intemperance, the aspect of the patient, the evidence of other effects of over-indulgence in alcohol, such as peripheral neuritis, are all valuable aids; and, coming to the more directly hepatic symptoms, we have to bear in mind that in cirrhosis of the liver the jaundice is less deep than in malignant disease or obstruction to the common duct, but ascites is frequent in cirrhosis, rare in malignant disease of the liver, and unknown in obstruction to the common duct. Often, too, in cirrhosis the liver

Digitized by Google

can be felt quite easily, and the hardness and roughness characteristic of the disease can be made out; there are none of the large lumps characteristic of malignant disease, which can sometimes be felt to be umbilicated, and may occasionally be observed to become larger very rapidly from hæmorrhage into them. The liver in obstruction to the duct may be a considerable size, but it is smooth and not excessively hard.

But although it is easy thus to put the distinctions on paper, cases of great difficulty sometimes arise; indeed, a post-mortem examination, even after sections of the liver have been cut, may fail to settle the question, for an infiltrating schirrus of the liver very closely resembles a cirrhotic liver. Of course the undoubted discovery of a primary seat of malignant disease will settle the question, but it is not infrequently impossible during life to decide whether hæmatemesis is due to a growth of the stomach or to cirrhosis of the liver.

Strictly speaking, no distinction should have been drawn between malignant disease of the liver and obstruction to the duct as causes of jaundice, for nearly always the cause of the jaundice in malignant disease is pressure on the common duct of a gland in the transverse fissure which has become infiltrated with new growth, or pressure on the duct of the pancreas which is infiltrated by new growth; but for clinical purposes the distinction is important, inasmuch as the malignant disease is necessarily fatal, but the other obstructions to the duct are not. Is the patient suffering from malignant disease of his liver, from an impacted gall-stone, or from chronic pancreatitis? This is a common and important difficulty. Before going further perhaps I ought to say what is meant by chronic pancreatitis. We owe our knowledge of the condition to surgeons. When, owing to advances in surgery, it became possible to operate upon patients suffering from impacted gall-stones, it was found that the head of the pancreas was sometimes hard and enlarged; this was thought to be due to growth, but the subsequent history of these cases showed that this was not so, and therefore the not unnatural conclusion was reached that the head of the pancreas was indurated by chronic inflammation the result of irritation of the impacted gall-stone as it lay in the common bile-duct where that joins the pancreatic duct. Then, again, surgeons who explored the liver with a view of discovering whether a case was one of malignant growth sometimes found the head of the pancreas hard and large even without a gall-stone being present; the gall-bladder was drained, the patient got well, and hence it was assumed that he had been suffering from chronic inflammation of the head of the pancreas. Later still it has been found that these patients will sometimes get quite well if left to themselves, and we can all probably recall cases which were condemned as suffering from malignant disease of the liver but which have got quite well, the jaundice slowly disappearing.

To return to the diagnosis of these cases of chronic jaundice. As a rule, if the jaundice is due to an impacted gall-stone, we get help in one of two directions: either the patient, who is usually a woman, describes attacks which point strongly to the passage of gall-stones on a previous occasion, or there are the rigors which point to infective cholangitis. An impacted gall-stone is usually doing little harm so long as the patient's temperature is normal. It must be remembered that probably very often cases of impacted gall-stone get well of themselves, for the stone becomes impacted in the wall of the duodenum where the duct passes obliquely through it, and from there ulcerates its way into the duodenum, is passed, and the patient recovers; but while, on the one hand, rigors with rise of temperature may be due solely to the presence of the stone, they frequently mean that either there is a condition of infective cholangitis due to the colon bacillus, or there is suppuration present which may take the form of suppuration in the bile-ducts, portal pyæmia with secondary abscesses in the liver, or an abscess around the stone. The temperature in all these cases of gall-stone is nearly always of the ague type—a rigor accompanied by a sudden excursion to a great height, say 104° or more, and then an equally sudden fall to normal or lower; indeed, it is well known that suppuration in connection with the liver is especially likely to be confounded with ague. There may be a rise of temperature in connection with both cirrhosis and growth of the liver, as was pointed out by Dr. Carrington; but then the temperature is rarely high, not exceeding 100° or 101°, and it is not of the remittent type. We may therefore conclude that it will only be difficult to diagnose an impacted gall-stone from other forms of obstruction of the common duct, or from malig-

Digitized by Google

nant disease of the liver, in those cases in which there is no history of previous attacks of gall-stones and there are no rigors. Fortunately such cases are rare, but they undoubtedly occasionally occur.

Next we have to consider malignant disease of the liver; here, as already mentioned, the jaundice is usually due to pressure of enlarged portal glands. In many cases the diagnosis is quite easy; the primary growth may be obvious in the rectum, uterus, or breast. I mention these three in particular, for I have known each of them overlooked. We may feel a large carcinomatous mass in the abdomen. In other cases, although the primary seat cannot be detected, other secondary growths—for example, in the lungs—may be discovered, and then the diagnosis is easy; or, again, the liver may be so enormous and irregular that there can be no doubt. It should be remembered that there is no disease of the liver in which the organ becomes so enlarged as in malignant disease; it may weigh as much as 12 lbs., and so much of the organ is destroyed by these huge masses of growth that we wonder whether a liver is necessary for life.

If we are in doubt whether a case is one of malignant disease of the head of the pancreas, some of the same principles will apply, for we may discover secondary deposits in other organs, *e.g.* the lungs; or the mass in the head of the pancreas may be so large that it must be due to growth, but this is exceptional. In the majority of cases, carcinoma of the head of the pancreas cannot be made out during life by palpation. This is partly owing to the fact that the secondary deposits in the liver make this organ big, and hence we cannot feel the subjacent pancreas.

There are one or two points which apply equally to the malignant disease of the liver and pancreas, but which are noteworthy. We must not place much reliance on pain; in malignant disease of both these organs it is frequently absent, or at any rate not severe. With regard to wasting, if this is very great it is some evidence of malignant disease, but patients with gall-stones may certainly waste, no doubt because of the anorexia and indigestion from which they suffer; and I have seen a patient with chronic pancreatitis wasted very considerably. On the other hand, it is remarkable how frequently patients with malignant disease will put on weight for a time when they come under medical care, for then they are fed properly. We ought, I think, to be very chary of resigning a diagnosis of malignant disease because the patient gains a little weight. It takes many years to acquire much experience, but my present belief is that a very deep olive-green-coloured jaundice is in favour of growth, and the dark olive green, skeleton-like patient, with his dry skin hanging loosely on him, who lies before his death perfectly quiet, apparently taking no notice of anything around him, and whose breathing is so shallow, and whose pulse is so feeble that it is often difficult to say whether he is alive or dead, is, I believe, usually suffering from malignant disease.

The last of the conditions we are discussing is chronic pancreatitis. A striking feature in connection with this is that, although the patient may be desperately ill, the disease is rarely fatal. I have been through our post-mortem records at Guy's Hospital for the past forty-one years, during which time there have been nearly 20,000 post-mortem examinations; there was only one certain case of fatal chronic pancreatitis, and there were only three others which could by any possibility have been examples of this disease. Therefore, if the patient is on the point of death, it is excessively unlikely that he is suffering from chronic pancreatitis. Not that this experience of the post-mortem room proves the disease to be extremely rare; probably many cases exist, but they recover even when desperately ill. Nor is the diagnosis impossible, for there are cases of chronic jaundice in which the evidence against cirrhosis, gall-stones, and malignant disease of the liver and pancreas is so strong that we may with fair probability urge that chronic pancreatitis is present. The various symptoms of bile circulating in the blood, and those due to absence of bile from the bowel, will obviously not help us to a diagnosis of the cause of obstruction, and sugar in the urine need not appear unless the pancreatic disease is very extensive. Fatty stools have been recognised, and probably correctly, as an indication of pancreatic disease, but they are certainly very rarely seen.

The next point to be considered is the treatment of these conditions; and we may take cirrhosis first. It is to be borne in mind that there may be a well-marked degree of cirrhosis without any symptoms due to it. Cirrhosis of the liver is from time to time found during routine examination, and a great number of cases met with in the post-mortem room have not shown any symptoms of cirrhosis

Digitized by Google

during life, just as there are many persons with granular kidneys whose disease gives rise to no trouble. The treatment of such a case of cirrhosis as this is merely to insist upon total abstinence from alcoholic drink; and this, too, is necessary when the disease is producing symptoms. Inasmuch as in many cases of cirrhosis the portal system is very congested, moderate hæmatemesis is salutary, and therefore needs no treatment; and in the same way bleeding from the bowels is rarely harmful, and piles are usually best left alone. In the rare cases in which hæmatemesis is dangerous, it is best checked by making the patient swallow a drachm of Liquor Ferri Perchloridi with an equal amount of glycerine; if this fails adrenalin may be tried. If there is ascites, the best drug to aid the removal of the fluid is copaiba resin; about 20 grains may be given for a dose; it is very nasty, and must of course be suspended, but it is a very efficient diuretic in these cases. In uncomplicated cases of cirrhosis the supervention of severe ascites means that the patient is near his end, and if the ascites is such as to need tapping, then probably the patient will not live longer than two months, and hardly long enough to need a second tapping; hence we learn that the beginning of tapping is the beginning of the end. From time to time cases are published which seem to point in a contrary direction, but, as Dr. Campbell Thompson and others have shown, these are almost certainly not cases of uncomplicated cirrhosis, but are suffering in addition from chronic peritonitis. This being so, it is not surprising that the operation of attaching the omentum to the abdominal wall, so as to try and short-circuit some of the portal blood into the general circulation, has not become popular. The patient with cirrhosis requires only the simplest food, and it is very necessary to keep the bowels open. For this purpose nothing is better than some calomel at night followed by a saline aperient in the morning.

In every case of gall-stones the first thing to be decided is whether or not to ask a surgeon to operate. Operation is imperative when jaundice with repeated rigors and attacks of pyrexia render infective cholangitis likely, for such a condition may easily become suppurative or lead to portal pyæmia; but I have known a patient so ill that a well-known surgeon and I decided she was too ill for operation, but she recovered completely without operation. Doubtless the stone ulcerated in the duodenum. Operation, too, is imperative if there is suppuration round a stone. But in the great majority of cases the indication for operation is not imperative, and much thought has to be applied to each case. The following points must be borne in mind:—Operations for gall-stones may be very difficult, and they should not be undertaken by any other than skilful surgeons. In the next place there is no doubt that people whose bile-passages are constantly being irritated by gall-stones are more liable to hepatic carcinoma than others, but although this extra liability is a point in favour of operation it is by no means so great as to compel operation in every case. And lastly, the social position of the sufferer makes much difference. If he is a member of the working classes and frequent attacks of biliary colic mean a loss of money, on this ground alone operation may be necessary. Gall-stones in the gall-bladder frequently produce local peritonitis around the gall-bladder, and that may lead to much pain from the formation of adhesions which may even constrict the duodenum and lead to gastric dilatation; and lastly, we must remember that the tendency to the formation of gall-stones ceases as people grow older; it is rare to find them active in people over seventy. I think there is no doubt that medical art can do much to relieve the sufferers from gall-stones, quite apart from operation. In the first place, morphia and a hot bath will relieve the immediate pain. But we must do more than this; we must try to prevent its recurrence. There is no doubt that tight lacing must be given up, for it displaces the liver downwards and in such a way as to hamper the gall-bladder so that the flow of bile is impeded; further, such tight lacing impairs the respiratory movements, which probably greatly aid the flow of bile. Gentle systematic exercise is useful, for it not only helps the general health, but increases respiratory movement. Horseback exercise has an especial reputation for these cases. The most divergent views have been expressed as to the influence of diet, but we do not yet know any article of food that encourages the production of gall-stones, and all that is necessary is that food should be small in quantity and easily digestible. All experience indicates that it is very desirable to keep the bowels open, and that this is best done with salines. These are commonly given as a

Digitized by Google

draught of any of the sodium and magnesium sulphate natural mineral waters, such as Pulna, Friedrichshall, Apenta, Hunyadi Janos, Æsculap, etc. There is no doubt that patients who are liable to attacks of gall-stone colic are very much benefited by periodic visits to Carlsbad and Marienbad, both of which have natural saline aperient waters. The patients are made to drink the waters and are put upon a restricted diet. But often for some reason or another patients cannot go to Carlsbad or any similar place, and then the best thing is to make them take large quantities of olive oil. Gall-stones are to some extent soluble in olive oil at the temperature of the body, and it is believed that oleic acid is absorbed and excreted by the bile. Be this as it may, I could if time permitted read you notes of many cases that have been benefited by olive oil. No one pretends that it will cure all cases; how can we hope that it will benefit the common case in which a dilated gall-bladder is stuffed with fifty or one hundred gall-stones so tightly packed as to form almost a solid mass? The oil can do no harm; it can be stopped at any moment; and if immediate operation is not decided upon it should be given a trial. It is a good thing for the patient to take as much olive oil a day as he can, but he need not take more than half a pint a day. It should be taken with meals. The simplest plan is to drink it out of a wine-glass, and some patients can do this. Many are, however, nauseated, and then the oil may be taken with salad or mashed up with potatoes or fish. If it produces nausea however taken, oleate of sodium sold under the name of eunatrol may be given; about thirty grains a day should be taken, and it is best given as a pill. Usually when these large amounts of olive oil are taken an aperient is not necessary, but if there is the slightest tendency to constipation a saline mineral water should be given on rising in the morning.

There is, unfortunately, very little to say about the treatment of malignant disease of the liver. If there is pain, morphia is our great help. In the rare cases in which ascites is present it will usually be found that this is really due to malignant deposits in the peritoneum; they may bleed considerably, and if the abdomen is tapped the fluid drawn off may be blood-stained. Probably in such a case the tapping leads to fresh bleeding, and therefore it is well in ascites due to malignant disease to defer tapping as long as possible. It has been suggested that when the jaundice is very deep and dependent upon malignant disease the gall-bladder should be drained to diminish the jaundice, but usually these patients are so desperately ill that we shrink from advising an operation. The itching in these long-standing cases of jaundice is sometimes well-nigh intolerable. The best lotion to relieve it is, I think, one of carbolic acid, 1 in 40; other lotions that are useful are dilute prussic acid and water, or one of ten grains to the fluid ounce of bicarbonate of soda, or one made with liquor carbonis detergens. Constipation is often a great difficulty in malignant disease of the liver, and enemata may be necessary.

We now come to a very important question—namely, what shall we do for the patient with chronic pancreatitis? Many would unhesitatingly say that the gall-bladder must be drained, and not until then will the patient improve, but the following case will show that this is not so:

A man æt. .62, seen July 2nd, 1901, at Hampstead, with Dr. James Andrews and Dr. Collingwood Andrews. The patient is deeply jaundiced, he looks very ill, and he is not strong enough to sit up. He tells us that a year ago he began to suffer from abdominal pain, and he has had it on and off ever since. It occurs in paroxysms at intervals of seven or ten days. Lately it has been very severe. For the last six months he has had jaundice, the depth of which has varied from time to time. He is hardly ever sick; there have been no rigors and no rises of temperature. During his illness he has lost 2 st. in weight. A careful examination (including the rectum) of the whole of his body revealed nothing abnormal, but some tenderness over the head of the pancreas. We expressed the opinion that the patient was suffering from carcinoma of the head of the pancreas. The patient and his friends asked whether any operation could be performed which would relieve him. We urged strongly against operation, but the patient and his friends were determined that an exploratory operation should be performed to see if so serious a diagnosis was correct; accordingly we reluctantly gave consent, and Mr. Edmund Owen was asked to perform it. A week later he opened the abdomen, and found the head of the pancreas so large and hard that, as this was considered to

Digitized by Google

indicate that the patient was suffering from carcinoma of the head of the organ, the abdomen was closed. The bile-passages were not drained in any part. Four or five days after the operation the patient began to improve in general health, his pain began to leave him, and his jaundice to disappear. From this time he put on flesh. I saw him next on August 16th, 1901. He then looked quite well, he was not jaundiced, had gained much flesh; but I thought I could feel a distinct lump in the region of the head of the pancreas. I saw him next on September 6th, 1902. He then weighed 12 st. 1 lb.; he looked in excellent health; he was back at his work. Nothing abnormal could be felt in the abdomen. The urine contained neither albumen, bile, sugar, nor diacetic acid. I heard from him June 11th, 1903, and he reported, "I am as well as usual, and have been so since I came to see you about a year ago. I shall be sixty-five years of age in September."

This case teaches that it is not always essential for the cure of chronic pancreatitis that the bile should be diverted from its normal track, and suggests that in some cases this has been done unnecessarily. We all know of cases in which after laparatomy for divers conditions benefit has followed, although no operation has been performed upon the diseased organ. Such cases are always difficult to understand, and are always open to the interpretation—hard, indeed, sometimes to believe—that the patient would have got well if no operation had been performed. We have all probably met with rare cases of jaundice which certainly did not appear to be gall-stones, and which were regarded as malignant disease, but after months the jaundice has disappeared, although no operation has been performed, and the patient has got well. It is of no use to record such cases as instances of chronic pancreatitis that have recovered, for some of them may have been examples of an impacted gall-stone that has ulcerated its way into the intestine; but, on the other hand, some may have been instances of chronic pancreatitis (like my patient, in whom no stone could be felt) that have got well apart from treatment.

The following is probably an instance in point:—A man æt. 60, seen with Dr. Lacey, of Woolwich. He had a single attack of jaundice fifteen years ago without much pain; it lasted a fortnight, and from his account was probably due to catarrhal jaundice. Now for three months he has had jaundice and pain, the pain not being paroxysmal like that of gall-stones; he has lost much flesh and has got weak, his jaundice has varied in depth from time to time and has never been very deep, his liver is much enlarged, but no lumps can be felt on it. There is no evidence of any primary growth anywhere. The date of this consultation was November 27th, 1902. I heard from Dr. Lacey on August 24th, 1903, that the patient had remained much the same for some time and had then gradually got better, his jaundice disappearing slowly so that it had vanished about six months after the consultation. The patient himself wrote to me in September, 1903, to say he was perfectly well. But cases such as these are not the only evidence that chronic pancreatitis can get well. Before the condition was recognised, examples of it were always regarded at the bedside as instances of malignant disease of the liver or pancreas; but if the patient died, no secondary deposits were found, and the head of the pancreas was seen to be enlarged and hard. I examined in our post-mortem records at Guy's all the cases for forty years entered as cases of malignant disease of the pancreas without secondary deposits. In many of them sections were cut of the pancreas, and the outcome of the inquiry is to show, as I have already said, that during forty years we only had one certain fatal case of chronic pancreatitis, and even with the most liberal interpretation of the cases there had not been more than three. But as now chronic pancreatitis is met with fairly often, it is highly probable that more than three instances of it came into Guy's in forty years. We may therefore conclude that it is a disease which usually tends to get well if left to itself, and that it is only in very severe and protracted cases that it is necessary to drain the gall-bladder. This conclusion is one that is of importance beyond the subject of chronic pancreatitis. We have seen that the common causes of persistent jaundice are cirrhosis, malignant disease, impacted gall-stone, and chronic pancreatitis. We have seen that we can usually separate from these cirrhosis of the liver and impacted gall-stone. That being so, it has often been urged that, unless the proof of malignant disease is conclusive, all cases supposed to be either malignant disease or chronic pancreatitis should be submitted to operation to dis-

Digitized by Google

cover whether they are chronic pancreatitis, for if they are the gall-bladder must be drained. This argument falls to the ground if my contention holds that most cases of pancreatitis get well if left to themselves. This is important, for on all grounds it is well to avoid needless exploratory laparotomy, and probably in time it will—as regards jaundice—be restricted to those cases in which it is thought there may be a gall-stone the removal of which is desirable, and those extremely rare cases in which the chronic pancreatitis is sufficiently severe to make it necessary to drain the gall-bladder.

This, I think, finishes the common diseases of the liver, and diseases of the pancreas so far as they are liable to be confused at the bedside with diseases of the liver. I have restricted myself to the diseases common in this country, and therefore have not discussed hepatitis, abscess, and hydatid; want of time has prevented my saying anything about the effects beyond the liver of diseases of it, such as the adhesions and intestinal obstructions that follow gall-stones. I have purposely not said anything either about the falling of the liver that is so common in women, especially those who lace tightly and have weak abdominal walls. My reason has been that unless perhaps the surgeon attempts to fix it little can be done, and usually the discomfort is hardly enough to ask a surgeon to interfere. But it is very necessary to remember how frequently the liver is displaced downwards in women. I have often known such displacement mistaken for enlargement due to disease. Further, tight and narrow waistbands and tight corsets may so compress the liver as to form a deep furrow. Often this is so deep that the piece of liver at the lower margin of the furrow may, when felt through the abdominal wall, feel like a tumour independent of the liver, and give rise to great difficulties of diagnosis, as may also the tongue-shaped process of the right lobe which was described by Riedel.

Finally, gentlemen, I must apologise for the many imperfections of this address; it teems with them. I have only tried very briefly to mention common points that occur almost daily at the bedside.

Harley Street,
February 8th, 1904.

WITH DR. CAMPBELL THOMSON AT MIDDLESEX HOSPITAL.

CLINICAL STUDIES IN DISEASES OF THE NERVOUS SYSTEM.*

THE TROPHIC CHANGES IN LOCOMOTOR ATAXY.

GENTLEMEN,—To-day I am able to show you some examples of the principal trophic lesions which may arise during the course of locomotor ataxy.

Each of the three patients before you has a perforating ulcer, and if you will look at these carefully you will see several characteristic points about them. In appearance the sores are indolent

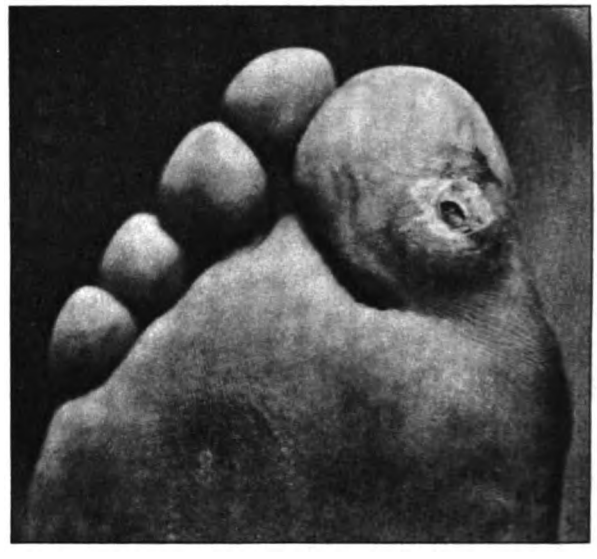


FIG. 1.—Perforating ulcer of the great toe in locomotor ataxy.

and dull, they show no tendency to heal, and at their edges the skin is thickened and raised to form a definite callosity. This inert condition seems to retard absorption, for, in spite of the little care the majority of patients take, it is rare to find any severe septic complications.

In one of these cases, however, the ulcer is more lively in appearance—its floor is reddened

* These "Studies" are from the teaching in the Out-patient Department of Middlesex Hospital.

Digitized by Google

and shows signs of granulating, and in fact presents much more the appearance of an ordinary sore which is slowly healing. This is not due to any spontaneous effort, but has resulted from treatment by the X rays to which it has been subjected. With this increased activity in the tissues came an increased power of absorption, and this patient recently showed some signs of local infection, which, however, quickly subsided under suitable treatment.

Another characteristic of these ulcers is absence of pain and tenderness. There is sometimes a certain amount of discomfort in walking, but not anything like the amount one would expect. This absence of pain can be well shown by probing the ulcer, and this probing also shows the depth of the ulcers, which you see is very considerable in the two cases which have not been treated; the crater-like mouths lead into a conical channel, which often extends right into the joints of the toes or feet.

You will also notice the position of these ulcers; they are all three on the plantar surface of the foot and all towards its fore part, this being the favourite position for them. The upper surfaces of the toes are sometimes affected, but the heels rarely.

There are several points of interest connected with the etiology of these ulcers. The underlying factor is no doubt an alteration in the nutrition of the part, while pressure and injury may fairly be reckoned among the exciting causes. Tourette has suggested that the ulcers originate from trophic changes in the small joints of the foot, and that the process arises from within outwards, and not from without inwards, being in fact a fistula leading from a diseased joint. Changes, of course, do occur in the bones and joints very often, and it is quite possible that the ulcers may sometimes arise in this way, but there must also be other factors present; otherwise one would expect such fistulæ to occur with the diseased joints that are so often found in other parts of the body in association with locomotor ataxy. The ulcers may sometimes heal spontaneously, but more usually need some definite form of treatment, and the scar in the foot of one of these patients indicates an old ulcer which healed some time ago after being scraped.

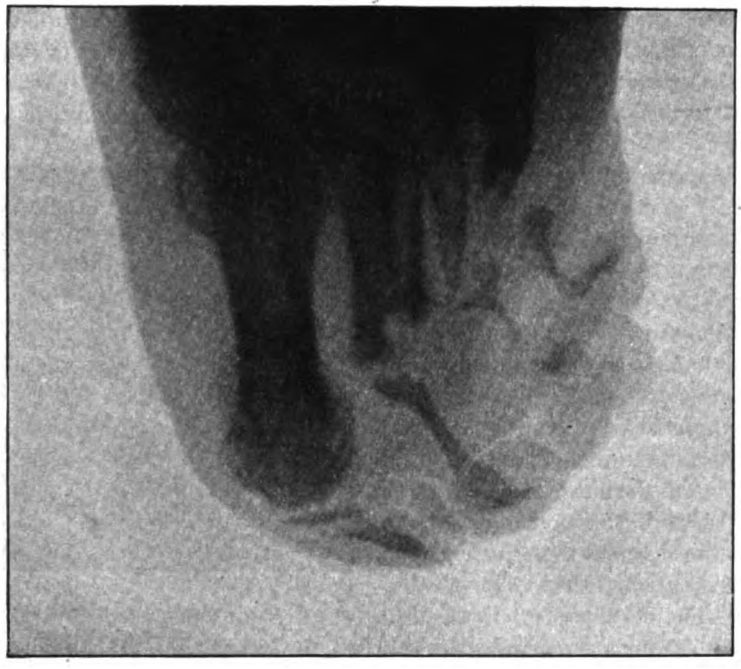


FIG. 2.—Skiagram of the right foot, showing great atrophy of the metatarsal and phalangeal bones in a case of locomotor ataxy.*

* This case is recorded by Mr. T. H. Kellock in the 'Clin. Soc. Trans.,' 1903, vol. xxxvi, and I am indebted to the Society for permission to make use of the illustration here.

Digitized by Google

Next there are the alterations in the bones and joints to be considered, the osteopathies and the arthropathies, and in the patients that I am now able to bring before you there are examples of both. You will have already noticed that in one of the cases of perforating ulcer the ankle-joint of the same foot is considerably swollen, and on closer inspection you will note that the swelling is firm though not very tense; in fact, it feels as if the soft tissues were swollen without there being very much free fluid in the joint. A skiagram of the joint, which I now pass round, does not reveal anything very definite, except that there appears to be some enlargement of the bone as well as of the soft tissues round about it. The joint is not painful, but of course considerably hampers the man's movements.

Now, if you will examine the foot of the next patient you will feel a considerable thickening round the metacarpo-phalangeal joint of the great toe, and we are told that this swelling was at one time much greater than it is at present. This is another example of the same kind of joint trouble. I will also ask you to observe another fact, viz. that the great toe of this patient's foot is shortened, and you will easily appreciate that by comparing it with the other foot. Normally the great toe should project beyond the others, but here it is distinctly shorter. (This shortening can be clearly seen in Fig. 1.) I have observed this shortening of the great toe in several cases, and have by the aid of skiagrams been able to arrive at its clinical significance. At first I thought it was probably due to some changes in the small joints of the toes causing the bones to be pressed more closely together, and that no doubt may sometimes be a cause, but the X rays show that the shortening in my cases is due to actual changes in the small bones themselves, although no such change is visible on examining the foot in the ordinary way. You will appreciate this point if you look at a skiagram of this case which I will now pass round. On comparing the pictures of the two feet you will see the obvious differences in length between the bones of the two great toes, while the fainter outlines of the soft tissues show the difference in length which you have already seen on the actual foot. Shortening of the great toe then would seem to be both of interest and importance, since it enables us to infer the probability of changes taking place in the bones themselves. It might perhaps be thought that the changes seen in the skiagram of this case are of no great practical importance to the patient, but this entirely depends upon the degree of atrophy, for if these bones were to get steadily smaller it would be a very serious thing, and that this does happen now and then will be seen from this other skiagram which I am enabled to show you of the feet of a patient recently under the care of Mr. T. H. Kellock, who has kindly allowed me to refer to the case. Here you see the bones are almost entirely gone, and as a result the foot is very much shortened and deformed (*v.* Fig. 2). The bones, when affected in locomotor ataxy, lose a large proportion of their lime-salts, become extremely brittle, and in the case of the long bones, often break under the least provocation, giving rise to the so-called "spontaneous fractures."

Fig. 3.—Wasting of muscle of hand in a case of locomotor ataxy.

I have next to show you a much rarer trophic change, viz. wasting of muscles. Tabes is essentially a disease of the sensory nerves, and muscular wasting is not often present, at any rate until the disease is far advanced. Sometimes, however, it does occur, and in the patient I now bring before you there is symmetrical wasting of the small muscles of both hands just like one sees in progressive muscular atrophy. There is some tendency to the claw hand, and the thumbs are rotated into the same plane as the fingers as the result of atrophy of the small muscles of the thumb.

The wasting has been present for more than three years, but fortunately it is not progressing. There is no anæsthesia, and the lesion is in all probability

Digitized by Google

due to a degeneration of cells in the anterior horns in the lower part of the cervical enlargement of the cord. The patient has all the characteristic signs of tabes, including loss of knee-jerks, lightning pains, Argyll-Robertson pupil, and ataxia. A little while ago he was under my care in the wards for a time, and was then treated by Frenkels method of re-educating the muscles to perform their movements again, and under this treatment he greatly improved. He now, as you see, walks with only quite a slight clumsiness, whereas formerly he was so ataxic that he was frequently looked upon as being intoxicated.

Queen Anne Street,
February 6th, 1904.

The Röntgen Rays as a Palliative in the Treatment of Cancer.—C. L. Leonard speaks of the unsatisfactory results following surgical removal of malignant growths. The rays may not cure any cases permanently, but they have shown a marked power to lessen pain, retard the progress of malignant disease, and add months and years to patients' lives. It is the duty of the physician so far as possible to palliate malignant disease, and this at least may be hoped for in the Röntgen treatment of inoperable cases. A few such have apparently been cured. The treatment of malignant disease by the Röntgen rays should be vigorous and as thorough as the vitality of the patient's normal tissues will permit. Anything short of this means failure. The dosage of the Röntgen rays is the principal factor; the border-line between injury to the pathological cells of low vitality and normal cells is not wide. If too little stimulation is used the cells of low vitality are not injured, and the normal cells do not receive the aid they need. Too great stimulation injures the normal tissue. An expert technique is as essential in the employment of the agent in therapeutic work as it is in diagnosis. The patient's reaction to the dose must be carefully studied. By a gradual course of treatment the skin can be tanned so that the dose can be increased almost indefinitely. The greatest care must be exercised in the case of large tumours not to cause a degeneration of too many cells, or the system may be flooded with toxins which it is unable to eliminate.—*Medicine*, January, 1904.

APPENDICECTOMY.

A discussion at the meeting of the Hunterian Society, held at the London Institution by

Mr. Harold L. Barnard	The London Hospital.
Mr. George Lenthal Cheatle	King's College Hospital.
Mr. James Berry	The Royal Free Hospital.
Mr. James Sherren	The London Hospital.
Dr. W. H. B. Brook	Lincoln Hospital.
Mr. Hugh Rigby	The London Hospital.
Mr. Michels	The German Hospital.
Dr. W. H. Best	Ilford.
Mr. Cuthbert Wallace	St. Thomas' Hospital.

DR. F. J. SMITH in the Chair.

MR. BARNARD, in opening the discussion, said: It is, of course, taken for granted throughout these remarks, that a thoroughly competent surgeon is available to perform the operations which are recommended. I think it is necessary to preface this, because if it is taken for granted that these operations may be performed by inexperienced persons, a good deal of harm will be done.

As to *how appendicectomy should be performed*, there appear to be two great dangers in the carrying out of this operation. One is always encountering ventral hernia following appendicectomy, and this is certainly a blot upon this otherwise extremely favourable and good operation. These cases of ventral hernia arise, I think, in nearly every case of appendicectomy owing to the method of splitting not being adopted. The old method of a two- or three-inch incision and cutting through the abdominal wall, regardless of the muscular fibres, until the peritoneum is reached, and then, having removed the appendix, suturing the abdominal wall regardless of its layers is responsible, I think, for a great number of cases of ventral hernia. Mr. Burney, was, I believe, the first man who introduced the splitting operation. It is a method universally adopted on the Continent and by many surgeons in London. The incision is at right angles to a line joining the anterior superior spine and the umbilicus. The external oblique fibres lie in the direction of the incision and are divided by the knife. The internal oblique fibres, which run in an exactly opposite direction, inwards and upwards towards the umbilicus, must be split. They are split by the points of two Spencer Wells forceps. The transversalis fibres are found to run transversely, and these are split and separated, and when they have been pulled

Digitized by Google

upwards and downwards the peritoneum can be opened in the direction of the original incision. When the operation is over and everything done inside the abdomen the retractors are withdrawn and the parts drop together into their normal position and are sutured up with catgut in the direction in which they were divided. The internal oblique and transversalis fibres are drawn together with the same catgut suture in a line from the anterior superior spine to the edge of the rectus; the external oblique fibres are sewn together again over these. There is then no weakness in the abdominal wall, and hernia is well-nigh impossible.

I now come to the risk of sepsis. It might appear that there is no greater risk in appendicectomy than in a radical cure of hernia. In a radical cure, if sepsis occurs it is the fault of the operator. But it is not equally clear that in appendicectomy it is the operator's fault because in the middle of his wound he cuts through the base of the appendix, and that little tube is of course a septic focus and is liable to infect the whole wound. We must recognise, therefore, that at the middle of the operation we have a septic focus appearing in our wound, and we must do our best to prevent it infecting the area and producing abscess, because then the risk of ventral hernia is very much greater, and even general peritonitis may supervene.

The method which I suggest as obviating this is as follows:—In the first place, it was Doyen who invented the angiotribe; he used it for the broad ligaments. He crushed the tissues so that they were smashed into a thin piece of parchment, and this he ligatured. He has used this instrument for the appendix. He smashes the base of the appendix, and the muscular coat and the mucous membrane are squeezed down into the cæcum. This is a great saving in the way of sepsis, and I think one should adopt this method, but not necessarily with an angiotribe. It is enough to place on the base of the appendix, when one has separated the mesentery, a pair of Spencer Wells forceps, and to place another pair as close as may be to these and slide the edge of the knife between the two. The mucous membrane is thus squeezed into the cæcum by the one forceps and into the appendix by the other, and the appendix may be removed without any septic matter escaping.

Then I think this division of the appendix should be done as late as possible. One should not bring a septic focus into the wound until the last moment. One should invaginate it and get rid of it as fast as possible. This invagination of the stump of the appendix into the cæcum need not occupy more than fifteen seconds if the purse-string suture is applied before the appendix is divided, so that the suture only has to be pulled tight when this is done.

There is another point with regard to the danger of sepsis in operations on the appendix. I do not think one should use silk more than is absolutely necessary in the performance of appendicectomy. A good many people use No. 3 or No. 4 silk, and it is no unusual thing to hear that after an operation performed by some leading man in the profession a sinus has formed, which has remained open for one, two, or three years, and after the lapse of much time from this has been discharged a silk ligature. This, of course, brings discredit upon an almost perfect operation. I think that catgut should always be used in the performance of appendicectomy. Should sepsis then happen to supervene the catgut will be absorbed in the course of a week or ten days, and one will not have to wait for a year or more for that piece of operative material to come away. So much for the technique of appendicectomy.

As to *when appendicectomy should be performed*, I think that we must have some facts, or what are as near to facts as we can get, with regard to this matter on which to base our argument. Therefore I have here put down in black and white, for the purpose of discussion, what appear to me to be the determining figures. *These are the facts* as far as I have been able to cull them from the literature and learn them from my own experience.

1. Taking the mortality of all cases, Sir Frederick Treves is of opinion that a common attack of appendicitis is attended by a risk of a 5 per cent. mortality. Some people think that too high.

2. In severe cases the mortality is much higher, as high as 10 or 15 or 20 per cent. These figures I derive from Dr. Hawkins, of St. Thomas's Hospital, who has published a most able treatise on appendicitis, and also from Dr. Fowler, of New York. That is the risk in hospital cases. The term severe which they use is, I think, a vague one.

3. Now what is the risk of taking an appendix out when a man has had an attack which has quieted

Digitized by Google

down? In the hands of ordinary surgeons it is less than a half per cent.; in the hands of Sir Frederick Treves it is 0·2 per cent. in 1000 cases.

4. As to the question of operation within the first twenty-four hours of the attack, this is an important point. An operation within that time appears to be attended—I have got only a general impression—by no greater risk than 5 per cent.

5. As to operation during the third, fourth, or fifth day of the attack, when lymph has been thrown out and the attack is in full blast, the mortality is given at as high a figure as 40 per cent. These are Dr. Fowler's figures.

6. Supposing the attack advances to a stage in which a man has a localised abscess—say, on the sixth or seventh or eighth day of the attack—what is the risk of opening the abscess? It is somewhere about 5 per cent. By some people it is given as high as 10 or 15 per cent. I have opened, I suppose, some fifty of these abscesses, and I do not think the risk is higher than one in twenty.

7. As to the mortality in general peritonitis, it is difficult to say what you are to call general peritonitis. Every case that dies is, of course, general peritonitis, but every case which is saved is not general peritonitis. I think you will find that the mortality from general peritonitis varies between 75 and 95 per cent., according to the operator. In thirteen cases recently published by Sonnenberg all died. Sonnenberg must regard only the very worst cases as general peritonitis, because most operators have a few cases which recover.

8. In cases of appendicitis which have got well, what is the risk for the future? When a person has got over one attack the question is often asked, "What is my risk for the future?" Apparently the risk of a relapse is one in three. I quote here Dr. Hawkins, who says one third of the cases at St. Thomas's Hospital relapsed within a period of three to five years.

9. How many cases fulminate, when do they fulminate, and what is the manner of fulmination? When a case of appendicitis fulminates it dies within three days. The greater number die between forty-eight hours and the end of the third day. These cases are 1 to 2 per cent. of all first attacks of appendicitis.

Having settled these figures, we now come to the question of when the appendix should be removed. This subject divides itself naturally according as to whether the appendix is removed (*A*) in the quiescent period, or (*B*) during the attack.

A. If we remove it *during the quiescent period* as a preventive against future attacks of appendicitis, then we may either remove it—

(1) After the second attack, or

(2) After a first attack, or

(3) After an attack of appendicular colic.

1. Sir Frederick Treves used to teach that the appendix should be removed after a second attack. I do not know whether he altered that opinion in the later days of his practice, but I remember when he used to teach that the appendix should be removed after a second attack. You know then that the patient is going to have "relapsing" appendicitis. It seems to me that whilst that is a very good method it is not an ideal one, because it is well known in the study of appendicitis that the first attack is the most dangerous one. In the first attack occur nearly all the abscesses, in the first attack occur nearly all the cases of fulminating appendicitis, so that any method which does not begin until the first attack is over and the second attack has come and gone, and then prevents the third, fourth, fifth, or sixth attacks, as the case may be, by the removal of the appendix, is not an ideal method. That is my objection to the operation in the interim period after the second attack.

2. Why not operate after the first attack? That I certainly think is the practical method. I think that anybody who has had a first attack of appendicitis should have his or her appendix out, when in such cases the mortality is only ½ per cent. when done by a competent surgeon. By having the appendix removed the patient will avoid the risk of several subsequent attacks and an average mortality of 5 per cent. at each attack. Patients avoid an enormous risk by having the appendix out after the first attack.

3. I do not know that the public at present are educated up to the point of permitting the surgeon to do more than remove the appendix after the first attack. The ideal thing which one has been able to do in only a few instances is to remove the appendix after appendicular colic. You will say, What is *appendicular colic?* It is attacks of pain due to the appendix and unat-

Digitized by Google

tended by fever. It is attended by hyperæsthesia. There are areas of referred pain on the anterior part of the abdomen, due to the distension of the appendix. Sometimes you can feel the appendix as a hard cord in the region of the right iliac fossa, which may or may not be tender. These attacks occur at frequent intervals. They sometimes last one hour, sometimes two; sometimes they last a little longer. They begin suddenly and end suddenly, and then the person is quite well again. These signs ought to enable one to recognise cases of appendicular colic. When one removes such an appendix—I have removed five or six of them—one finds that either the interior of the appendix is in a catarrhal or ulcerated condition, or there is stricture or kinking of the appendix, and an irritant secretion distending it. In many cases calculi are present. Sometimes these attacks occur in women in connection with menstruation. There is a little artery connecting the right ovary with the appendix, and lymphatics from the right ovary pass to the appendix, so that it is not surprising that there should be these attacks at the monthly period, especially if the appendix is already congested. These attacks are difficult to distinguish from true right ovarian pain. These, then, are the conditions which one finds in cases of appendicular colic when one removes the appendix.

If you want to know the relation of appendicular colic to appendicitis, we have first of all to recognise, as Sir Frederick Treves says, that appendicitis is a peritonitis in relation to the appendix. To that we may add that appendicular colic is due to any less extensive disease of that organ. The mucous membrane alone may be inflamed, or the muscular coat inflamed, or there may be simply a distension by fluid or fæcal concretions. I may add that in these cases in which the appendix was removed there has been a great improvement in health. This retention of secretion in the appendix has a distinctly bad effect on the health of the patient. The recurrent slight attacks were stopped by the appendicectomy, and the health of the patient greatly improved.

B. We now come to a much more debatable question—*appendicectomy during an acute attack.* I think it is well that we should divide it up under the following heads:

1. The first twenty-four hours of the first attack.
2. The first twenty-four hours of a subsequent attack.
3. During the second, third, and fourth day.
4. During the abscess stage.
5. When general peritonitis is present.

1. First of all, as to appendicectomy during *the first twenty-four hours of the first attack*, I think that is as ideal a method as one can advise in acute attacks. A person is going to have a first attack of appendicitis, he has all the symptoms of the disease; now if you can take his appendix out at the very beginning without unreasonable risk, you will of course avoid not only the risk of this but of all subsequent attacks. Moreover I think that appendicectomy should be done then, inasmuch as we shall thus avoid the risk of being compelled to operate during the second, third, fourth and fifth days, with a mortality of 40 per cent. I think that one can operate during the first twenty-four hours with a mortality of not more than about 5 per cent. I do not say the public are ready for it yet, I do not say it is practical, but where one has the opportunity one should certainly remove the appendix during the first twenty-four hours of the first attack. It is easily done at that period; there are no adhesions, there is very little difficulty in finding the appendix, and as a rule it is not perforated, and therefore infection of the peritoneum is slight. One can know whether the appendix is perforated or not by the absence or presence of hyperæsthesia, as has recently been demonstrated by my friend, Mr. James Sherren. If hyperæsthesia is present it is pretty certain that the appendix is not perforated, but if hyperæsthesia should disappear within the first twenty-four hours of the attack then perforation is certainly present, and this is all the more reason why the appendix should be removed, because if the appendix has perforated it has burst into the open peritoneal cavity, and the only chance then is to remove it. Moreover if you operate within the first twenty-four hours your peritoneal stitches will hold, but not afterwards. Having cut off the appendix within the first twenty-four hours of the attack, you can invaginate it into the cæcum, but later on the stitches cut out and the stump must be left unsequestered.

2. As to *the first twenty-four hours of a subsequent attack*, this is not quite so plain a case. In

Digitized by Google

many instances there may be adhesions from a first attack, so that the appendix is not so easy to find and it is more difficult to remove. The risk in a subsequent attack is not so great as in the first attack. Therefore the difficulty is greater and the advantage to be gained is less, so that I am not so keen on appendicectomy during the first twenty-four hours of a second or third attack as I am for appendicectomy during the first twenty-four hours of the first attack. I think that in cases of a second or third attack I should reserve appendicectomy for very acute cases.

3. In any attack of appendicitis I should be prepared to say that *appendicectomy* should not be done *during the second, third, or fourth day*, except as a forlorn hope or when one is pushed to it by the case becoming progressively worse. One's difficulties are these: when the abdomen is opened so much lymph is present that you cannot readily find the appendix. You have to rummage round and hunt for it. In doing this you break down adhesions, the lymph-barriers, which are shutting off the general peritoneal cavity from the acute process going on. You do more harm than good in doing that. In many cases the appendix is already perforated. The damage, as far as it can be, is already done, and the removal of the appendix at that stage is simply shutting the door of the stable after the horse has been stolen. Moreover when this operation is done with proper precautions you have to make a large incision, and so there is the greater danger of ventral hernia, and this means a miserable life ever afterwards. So I do not think, excepting under unusual circumstances and when one is pushed to it by the case progressively getting worse and worse, that one should even open the abdomen during the second, or third, or fourth, or fifth days. But during this stage, if one does open the abdomen one should simply drain and put a tube down as near as may be in the middle of the inflammatory area.

4. As to *the abscess stage*, where a case has run to six or seven or eight days and there is a large abscess, there is no doubt that it should be opened and drained. Of that every one is agreed. But I am very opposed to removing the appendix at this stage. It seems a gratuitous act to hunt in the midst of this bath of pus for an appendix which may be stowed away in any corner. The appendix is in such variable positions that you cannot know for certain where it is, and the man who hunts round for it is liable to break down all adhesions and let pus into the general peritoneal cavity. The incision must be very large, much too large for a splitting operation, and so once more ventral hernia is very likely. With regard to the question of risk, I do not think the operation can be done with less than 8 or 10 per cent. mortality. I had a letter from Mr. Rutherford Morison's assistant (Newcastle), and he assured me that Mr. Morison has done more than 100 consecutive cases without a death. But certainly the risk of ventral hernia must be very great in an incision which, in Mr. Morison's figures, is shown to be five or six inches long.

5. As to *cases of general peritonitis*, I do not know that it matters whether you take the appendix out or not as long as you do not waste much time over it. The appendix has burst and the whole peritoneal cavity is infected, and whether a man shuts the door or not after the horse is stolen does not matter very much, so that he wastes no time before pursuing and capturing the thief. So that I think if, in a case of general peritonitis, you find the appendix absolutely presenting in the wound, then you may tie a ligature round it and cut it off. But if it does not present I do not think you should waste any time in looking for it.

Mr. CHEATLE said: I do not intend to waste time in following Mr. Barnard through many of the practical points which he has so ably brought before us. Mr. Barnard has great fear of breaking through an abscess wall, and evidently treats its parts as essential to the safety of his patients. In a sense I agree with Mr. Barnard, and in a sense I do not agree with him. I quite see that an abscess wall, when it has formed and has allowed the accumulation of a considerable amount of pus within its cavity, is to be respected to a certain extent, and that it is not wise to allow the abscess to pour all its contents within the general peritoneal cavity. But I do not believe that after the evacuation of pus any harm is done by breaking through the remaining walls in looking for and removing an accessible appendix. The greatest argument against so doing is to me not the possibility of spreading infection; but the completeness with which an appendix is hidden often renders the search unfruitful, even when the appendix has

Digitized by Google

not been completely destroyed. As far as breaking the continuity of the abscess wall is concerned, its safety seems to me wrapt up in the mysterious process of immunity. Immunity must, to a great extent, have occurred before the vital processes involved in the formation of an abscess wall are possible. They could not begin and continue in the presence of a micro-organism whose products were still capable of inducing death and destruction on the tissues of its host. It seems to me that the pre-existing connective tissue-cells round a developing abscess have become so little affected by the products of infection that they are able to divide, multiply, and become vascularised, so as to encyst the focus and complicate the disease by allowing the collection and storing up of more products, which, had it not been for the encapsulation, would have been absorbed by the lymphatics before a great collection were possible.

Hence the abscess wall requires a surgeon's knife before the case can get well, and then it gets well not because the surgeon has not interfered with the abscess wall, but because the case has become immune to its infective process. I have frequently opened perityphlitic abscess cavities through apparently normal and unaffected abdominal walls, but I have never seen a spreading infective process occur therein, even though their wounds were covered by highly virulent micro-organisms during the evacuation of the pus. There was no pre-existing abscess wall to protect these wounds. I believe a surgeon would be much more accurate if he considered whether the patient had become immune rather than whether the abscess had become encysted or not, and if necessary wait for the immunity, which is shown by the complication of the disease by the formation of adhesions and abscess walls rather than the encystment of the pus. I was led to think the abscess wall a less sacred structure after the following experiments.

I inoculated the subcutaneous tissues of guinea-pigs with *Staphylococcus pyogenes aureus* in sufficient doses to produce acute abscesses, and killed them at different stages in the progress of disease. At the end of the first ten minutes I killed several guinea-pigs, and frequently found staphylococci in the livers and spleens. So that even in the early stage one saw that probably a generalised infection had been conquered before the typical localised acute abscesses were produced. One hour after inoculation I saw death in various tissues. Around these dead areas were living areas, around the margins of which had collected many leucocytes. At the end of seven days I saw a well-marked abscess wall, and in the wall many micrococci. These were virulent to other guinea-pigs. I could not see any cocci outside the wall. I took away those micrococci and inoculated them into the same guinea-pigs, on the whole with little effect. Again, the pus in the abscess did not quite reach the abscess wall in many places, and in many cases the abscess wall was incomplete. From these observations I inferred that protection was more a question of local or general immunity than a mere mechanical obstruction to the escape of pus due to the presence of an abscess wall.

(*To be concluded.*)

Crude Petroleum.—Binkerd has used the finest grade of crude petroleum, and with the best results, in all forms of acute affections of the respiratory tract. It relieves the laboured and stridulous breathing, cyanosis disappears and expectoration is facilitated, the fever subsides, and the patient goes on to convalescence and prompt recovery. He experimented on himself when in bad health from malassimilation, hæmorrhoids, and rectal prolapse, using crude petroleum, and found that it agreed with the stomach, increased the appetite, caused gastric fermentation to cease, and regulated the bowels and kidneys. After two years' daily use of crude petroleum no distaste for the oil has developed; one can take it as readily as milk or water. He does not consider it a food, a tonic, nor yet a stimulant, but he thinks it is a complete intestinal antiseptic and disinfectant, and it has demonstrated to his full satisfaction that it has an anti-fermentative action. He twice purposely crammed himself with indigestibles at supper-time, following with petroleum, and had no bad effects, except a large evacuation of the bowels and an eager appetite for breakfast. Recently he has been employing it in the treatment of typhoid fever in half-teaspoonful doses every four hours with highly satisfactory results. He thinks it is entirely safe; it is abundant and cheap, and as it is not changed by contact with the intestinal secretions, it is not incompatible with any other remedy that may be advised.—*Therapeutic Gazette; Journ. A.M.A*, January 16th, 1904.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 590. WEDNESDAY, FEBRUARY 17, 1904. Vol. XXIII. No. 18.

CONTENTS.

PAGE

*A Clinical Lecture on the Diagnosis and Treatment of Hæmorrhoids. By A. MARMADUKE SHEILD, M.B., F.R.C.S. 273

*Appendicectomy. A DISCUSSION BY H. L. BARNARD, G. L. CHEATLE, JAMES BERRY, J. SHERREN, W. H. B. BROOK, H. RIGBY, A. MICHELS, W. H. BEST, and C. WALLACE (*concluded*) 282

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE

ON

THE DIAGNOSIS AND TREATMENT OF HÆMORRHOIDS.

Delivered at St. George's Hospital.

By A. MARMADUKE SHEILD, M.B., F.R.C.S.

GENTLEMEN,—I have been asked to-day to speak about hæmorrhoids and their treatment. You have to remember that "the piles" is considered by many of the public to be practically the only disease which is found about the anus. This is the "general" term for any disease of the rectum, and it does not do, therefore, to take the patient's word about what he is suffering from. Please recollect with the greatest care, when a patient, even an intelligent man, comes to you and says "he is suffering from piles," it does not necessarily follow that his opinion is right. A second point—and it is a very peculiar one—is, that medical men themselves, when they suffer from any rectal disease, generally think they are the victims of cancer. You will find one nervous doctor with "a fissure" will come and say he is suffering from cancer of the rectum! Another will come with piles, and another with a small fistula, saying all is over with him, and he is the victim of malignant growth. Thus, among members of the public everything in the anal region is "piles," and in the case of medical men themselves they fear all rectal disorders are cancer. Unfortunately doctors make mistakes in this direction in two ways: they overlook cancer in their patients, and imagine that they have cancer themselves when they have not got it!

First of all, to clear the ground, we will start with a definition of what hæmorrhoids really are. Piles are nothing more or less than a pouched varicose condition of the veins of the rectum about

Digitized by Google

its lower end. The various changes which occur in varicose veins—namely, thrombosis, clotting, inflammation, thickening and organisation of tissue, the formation even of fibrous or calcareous masses in the veins, in very rare cases, of suppuration—are all here found. Then, above all things, these varicose veins are apt to ulcerate and bleed. There is no difference in the pathology of piles and varicose veins in the legs, and the changes which occur in them are precisely the same even to thickening and hypertrophy of the skin and cellular tissue, and the existence of eczema and fissures. Piles are usually divided for purposes of description into those which are external to the rectum—that is to say, around the margin of the anus,—and internal piles, which occur covered by mucous membrane. But in practice there is a third variety, which is perhaps more common than either, and that is the so-called mixed pile, partially inside and partially outside the anus. There is an essential difference in the structure of external piles and internal piles. The external piles are covered by skin almost entirely, and are venous in character. Internal piles are covered by mucous membrane and are far more vascular than external piles, and they are said by many authors to contain arteries. That is a disputed point which I shall not enter into now. But the broad clinical fact which you have to remember regarding the structure of internal piles is that they are cavernous in structure, exceedingly vascular, and far more difficult to treat than the external form. You know, or at any rate those of you who have just done your anatomy will remember, that the hæmorrhoidal veins form a dense plexus round the rectum unsupported by firm tissue, and that they are destitute of valves. And those of you who have just passed your anatomical examinations will recollect that they empty themselves in two directions: into the internal iliac vein radicles; straight through the portal system into the liver. There are a number of reasons why the veins at the margin of the anus should become varicose. They are, as I have said, not supported by firm tissue and are destitute of valves, and consequently in a condition which will readily render them prone to dilatation.

Now the causes of varicosity of the veins of the rectum, or piles, will next engage our attention. One of the first and most important causes is mechanical pressure, just as with varicose veins in the legs. And the mechanical pressure which produces piles is very various. If I were asked the leading cause of piles I should say chronic constipation and the passage of hard fæces. Chronic constipation in the production of piles acts in three different ways. In the first place, there is the mechanical pressure of the fæces on the rectal veins; secondly, there is the constant irritation and congestion, and the inflammation caused by the presence of the fæcal lump; and thirdly, there is the straining to overcome the fæcal obstruction, which is a very powerful factor in causing mechanical congestion of the veins of the rectum. Remember these different ways in which chronic constipation tends to the production of piles. Other familiar and important clinical causes of piles are the pressure of the gravid uterus, large fibroids of the uterus, and malignant growths of the uterus, or of the prostate or bladder, or other parts inside the abdomen. Next we have those conditions of the liver which obstruct the portal circulation. And, of course, immediately we begin to think of hepatic congestion, of cirrhosis of the liver, and of cancerous and other growths of that organ. Going still further back, you have to remember that some of the worst cases of piles which are met with are those associated with the obstruction found in mitral valve disease. That affection known as "nutmeg liver" is another condition which is found in association with heart disease and hæmorrhoids. Any of you who frequent the post-mortem room will know how in obstructive mitral disease one of the last changes in the case is the occurrence of a "nutmeg liver," with an exudation of dropsical fluid into the abdomen, and portal congestion, and consequently piles. Having said so much, you will see that before you begin to talk cheerfully about operating upon a case of piles there are many things to be considered; and any really sensible doctor will investigate the general medical condition of his patient very carefully before he acts. I have already mentioned to you, just now, what an important mechanical cause of the production of piles is "straining." This is especially found to be true in chronic constipation; and it is also found in association with bladder diseases. Thus you will find that piles is a not infrequent accompaniment of enlarged prostate and stone in the

Digitized by Google

bladder. You will often find a man who has a large stone suffers from very bad piles. Such considerations are very important, as you will see when we come to talk of the treatment of the disease.

I have mentioned to you the structure and causation of piles, and now we will pass on to the appearance of them. First of all let us speak of external piles, for an inspection of them is concomitant with their diagnosis. Anybody who has had the least experience of rectal cases—and you will see plenty of them in the out-patient room here—will know the small livid or bluish tumours which appear around the margin of the anus. In fact, there are only two things which external piles can be confounded with. The most common condition with which they are confounded is that of syphilitic condylomata. That which they are least often confounded with is sebaceous cyst at the margin of the anus. A case of sebaceous cyst in the skin of the ischio-rectal fossa is a favourite "examination case." The careless candidate, thinking nothing else but piles could occur in such a situation, is "trapped." The diagnosis of condylomata from piles is made at a glance by those who have seen both these disorders. The white sodden appearance of condylomata, and the discharge with which they are covered, is familiar to anyone who has seen the affection. Indeed, the mistake between external piles and condylomata is most frequently made by those medical men who persist in taking the statements of patients who come to them for advice as gospel, instead of looking and judging for themselves. A young man will walk into your room and say he has got piles, and that they pain him a good deal. You are busy and tired, and you give him that familiar but very inefficacious gall and opium ointment, and the young man walks away satisfied, with a syphilitic rash all over his body and syphilitic condylomata round his anus. When external piles become inflamed they are very apt to become thrombosed. A thrombotic external pile is apt to be red, hot, hard, and painful, and it is for this pain and heat that the patient usually consults the doctor. When the inflammatory action subsides the clot becomes what is termed "organised,"—that is to say, transformed into connective or fibrous tissue. Thus are formed those hypertrophied tabs of skin which are so often seen about the anus in conjunction with external piles. I now want to mention a matter in connection with external piles of the very first importance in practice. These little swellings are commonly associated with eczema and violent irritation or itching at the margins of the anus, and very frequently they are associated with fissure. There is a well-known form of rectal disease known as "fissure," a painful "crack at the posterior margin of the anus. And this "fissure" is often covered over by a small pile, which has been well termed by authors a "sentinel pile." No mistake is more common than to overlook this little fissure which lies at the base of the pile which is on the coccygeal aspect of the anus. If you remember this you will certainly not, I think, make the mistake so often committed—namely, to remove the pile and leave the fissure untreated and unnoticed. When I say that external piles are commonly associated with excoriations and cracks and eczema about the anus, I should also mention that there may be also commonly associated with them "pruritus ani." You know the troublesome disorder which goes by this name, and which the books describe as being inveterate to treatment, sometimes even causing the patient "to renounce the pleasures and duties of society." This distressing malady is often associated with some local cause, and this is often a "tab" of skin, a pile, or a "fissure." One of the first essentials in the treatment of pruritus ani is to be on the look-out for these little flaps of skin and fissures, and treat them before you treat the pruritus by lotions or ointments. So much for external piles; and perhaps the most important point connected with the subject is not to overlook fissures and eczema associated with them, nor yet to overlook other diseases—such, for instance, as cancer. It is a very common thing indeed for a cancerous ulceration to exist high up in the rectum, and for the patient to also have external piles. In such a case a rash or inexperienced man may put the patient under an anæsthetic and operate upon the external piles, and at the same time absolutely overlook the cancer higher up. He would, therefore, be committing a grave surgical error, an error which could have been avoided by carefully examining the rectum before operating. Before any operation on external piles is attempted the rectum should be carefully emptied by enemata, and then a painstaking examination should be made to make sure that there is no serious disease above complicating the disease which you see below.

Digitized by Google

The treatment of external piles, as of internal piles, resolves itself into general and local, and you must never neglect the one for the other. Before you think of operating for external piles you will carefully inquire into the patient's condition. If he be, as these people often are, a plethoric, full-blooded man, much addicted to dining in the City and taking large quantities of alcohol, you will probably find that regulation of diet, cutting off his alcohol, and the administration of those old and valuable remedies blue pill and purgative saline draughts, will probably cure him without operation at all. These remedies will work wonders in cases of external piles and rectal congestion in plethoric, hard-drinking, over-eating men. This important branch of treatment must never be neglected in such cases. Suppose you have regulated the patient's diet and purged him, and he still suffers from irritation about the anus, the treatment of external piles surgically is sufficiently simple. The rectum is emptied by means of an enema, the parts are carefully cleaned, and the piles simply removed by snipping them away. If they are of considerable size, it is a very good plan to turn down two little short flaps of skin at the base of the pile, snip it away, and sew the pieces of skin together with fine catgut; this is a favourite operation of mine. The one "pitfall" in this operation is the too free removal of the skin. If, as is sometimes done by a "free operator," the external piles are cut away with all the skin at their bases, you get a most unfortunate result—namely, contraction of the anus. If you cause this to a patient it is one of the worst things you can do. The contracted parts have constantly to be dilated by bougies, or freely divided, and you will know to your chagrin that a patient otherwise in good health is spending his spare time in advising his friends to avoid you as a surgeon in future. Thus an adage of mine is well exemplified—that what is called "minor surgery" often leads to "major complications."

What shall be the treatment of external piles when they are inflamed and thrombosed? It is then the patients generally come to you for relief. In this matter authorities differ widely. The ordinary practice is to transfix the inflamed pile and turn out the clot. Or you may remove it altogether. My own advice in these cases is to avoid operation if you can until the inflammation subsides. The old rule of not operating upon inflamed parts is still a useful one. I think in such cases, if you purge the patients freely and give them sedative lotions, such as iced lead and opium lotion, to bathe the anus, and let them rest in bed, probably the operation does better after the acute inflammation has subsided. But that is only my own opinion. Other surgeons think the opposite, and I do not know that it matters very much.

So you see there are a great many little points about the treatment of external piles, and about their recognition, especially that little matter of overlooking a fissure of the anus and the "sentinel pile" which covers it.

Now when you come to hæmorrhoids inside the rectum you have to consider a far more serious matter. I have a very good specimen of the disease here. You see the formations are situated well within the external sphincter, and are much more vascular than external piles. They consequently bleed very much more freely, so that one of the leading symptoms which a patient will complain of is loss of blood on going to the closet. There is one peculiarity about the bleeding of an internal pile which is useful as a clinical hint. The hæmorrhage is almost always arterial in character, and spurts, so that the blood will invariably splash or splatter upon the pan of the closet. This kind of bleeding is almost diagnostic of internal piles. In the case of bleeding from cancer, in the vast majority of instances, the blood is mixed with slime and diarrhœa-like evacuations, and is much more intimately mixed with the motion than in the case of piles. Some patients, especially women, are naturally reticent about rectal symptoms, so remember that the only symptom of internal piles may be excessive anæmia. The mistakes made in practice in these cases are almost incredible. I have known them diagnosed as "pernicious anæmia," have known them repeatedly to be treated with large doses of iron, which increases the constipation and makes the bleeding worse! "Changes of air" and expensive foreign spas have also been before now prescribed for a patient who is daily bleeding to death. I beg your close attention to this part of the subject. The next symptom which these patients suffer from besides loss of blood is a constant source of irritation and weight about

Digitized by Google

the rectum. They complain that after the bowels have acted masses come down outside, as they express it, and they have to put them back with their fingers, and undergo much trouble and misery. Sometimes these piles, as with external piles, become inflamed, and they not infrequently become protruded and strangulated. That is a most awkward business to manage. You find a nervous old lady or old gentleman with internal piles protruded outside the anus, of a dark plum-colour, suffering agonising pain. The treatment of these cases is often very difficult indeed. One of the best ways of managing them is to place a patient upon her face, with the pelvis well raised by pillows, and after applying warm opiate lotions to the parts and allowing a period of rest you may attempt to return the piles, gently, within the sphincter by pressure with your finger. But if you fail in that, it is far better, according to most authorities, to administer an anæsthetic, dilate the sphincter, and proceed with the removal of the piles at once. If the patient refuses the anæsthetic or the operation, all you can do is to keep the pelvis raised and the parts bathed with opium lotions. Frequently the masses will slough off and cure themselves, but this is a long and painful process. My own opinion is in favour of a radical operation in these cases.

Now comes the most important part of this lecture, and that is the method of examining the rectum in a case which you suspect to be one of internal piles. If you fall into that common error in practice of accepting patients' opinions of their own diseases, you may make some of the worst mistakes possible. As an illustration of how disastrous these errors may turn out to be, let me tell you the following:—I remember a few years ago a young lady, not much more than twenty years of age, who went to her doctor and complained that she was suffering from piles, and that she passed blood from them at stool. Thus were the usual symptoms of piles described. The doctor concluded without examination what was the matter with her, and he gave her the usual gall and opium ointment. Shortly after this she became engaged to be married, and her mother, who was a sensible woman, was a little bit afraid of these piles, and asked the doctor whether it would make any difference to her daughter's matrimonial prospects. He gave her some more ointment and said, "No, it would not make any difference," and that the use of the ointment would get her well. The young girl was married, and after marriage her symptoms became worse, and there was more pain, bleeding, and irritation. It was under those circumstances she was brought to me. I at once suggested a local examination, which was met by the usual refusal. I made the reply that I was sorry I could not prescribe for the case or pronounce an opinion upon it. The patient left the house, and, as generally happens under those circumstances, she came back and allowed an examination to be made, and it disclosed a very bad state of things. This unhappy young woman was found to have advanced cancer of the rectum, and, worse than that, she bore in her uterus a fœtus of three or four months' growth. Here, therefore, was a case of combined pregnancy and advanced cancer of the rectum. Probably some of you would scarcely have believed that so young a patient could have cancer of the bowel, but I assure you that they do, and I have several times seen cancer of the rectum before the age of thirty. Therefore recollect, when a patient comes suffering from symptoms of piles, do not neglect to insist upon a local examination. The right thing to do when a patient comes complaining of these symptoms is to put him on his side and give him a copious enema. My practice is to throw a pint of warm water slowly into the rectum, allow it to remain there for ten minutes, and then let the patient go to the water-closet and evacuate it. This means that the rectum is emptied and cleared for the examining finger, and it is certainly much more cleanly for the surgeon. If any piles are present they protrude through the sphincters and come readily into sight as florid bleeding masses, once seen never to be forgotten. Thus the first essential in making a rectal examination is to give the patient an enema. Of course, this is seldom done in hospital practice. The pressure of work in the out-patient department precludes this method being always pursued. But the examining with the finger a rectum which is loaded with fæces is as improper as it is repulsive to the surgeon. Nothing is more likely to lead to diagnostic errors than this practice.

Having made the diagnosis of internal piles, the next question which occurs is that of treatment. There are very few cases of internal piles which

Digitized by Google

cannot be operated upon; but there are exceptions. For instance, it is of little use operating upon internal piles in a woman who is advanced in pregnancy. I have seen a woman with bleeding piles about the seventh or eighth month of pregnancy submitted to an operation, and I have seen an abortion in consequence, and the woman made dangerously ill. Do not operate for piles upon a woman who is advanced in pregnancy. And do not operate for bleeding piles if she has an enormous fibroid of the uterus. Such a tumour would press upon the iliac veins, and in such a case it would probably be the right course to remove the fibroid. Neither, as a rule, should you operate upon piles when they are associated with advanced disease of the liver, as cirrhosis. Should you get a poor man lying in bed with a "nutmeg liver," with heart disease, and losing blood by the rectum, it is very questionable whether he should be operated upon. At all events, a wise doctor will consider such a case as this eminently one for consultation. There is a third class of case in which it is not always wise to operate for piles, and that is in exceedingly plethoric, "thick-necked," full-blooded people. There is no question that some of these individuals engorge their livers with rich food and large quantities of wine, and occasional bleeding from the anus is a safeguard to their general circulation, and the occurrence of such bleeding may possibly ward off the onset of apoplexy. I do not say such patients should never be operated upon, but recollect you must always consider very carefully whether you ought to do it or not. There are many cases where there is no doubt operation should be done. Such are instances of pronounced anæmia from loss of blood, and great irritation about the rectum. A patient who has perhaps lost blood for fifteen years with his stools, is anæmic, pulled down in health, always suffering with misery about the rectum. The blood even runs down inside his trousers; the piles are constantly protruding, and have to be replaced with the fingers. These are the cases which urgently need operative treatment.

The operations for internal piles are very numerous; and if you are writing a paper in examination you will have to mention a long list. There is firstly, operation by ligature; secondly, operation by clamp and cautery; thirdly, operation by crushing the pile; fourth, injection of the pile; fifth, puncture; and lastly, electrolysis. And, to finish up, there is excision of the piles and venous area of the lower rectum, the so-called Whitehead's operation. These are all operations in vogue for removing piles. A large number of them I think had better never be performed, but it is my duty in a lecture to tell you what I know about them. I will now speak of the operations which I do not personally very strongly approve of. First of all, Whitehead's method. This operation is very largely practised by its distinguished inventor and a number of surgeons in London and in the provinces. It consists, as you know, in making an incision round the margin of the anus, at the muco-cutaneous junction, reflecting the mucous membrane and dissecting away the whole of the veins and hæmorrhoids, tying the vessels, and afterwards sewing the mucous membrane to the skin. This operation, though surgically sound, has these possible disadvantages. It is a little difficult of performance, it takes a long time, and it is sometimes associated, even when done by the best surgeons, with serious contraction of the anus afterwards. There is no getting out of this fact. Patients have come to consult me with this troublesome contraction of the anus after the performance of this operation by some exceedingly good operators in London and the provinces. This is enough to prevent me from recommending it to you as an operation to be generally performed. Next with regard to such proceedings as electrolysis, ignipuncture, and injections of piles with carbolic acid. I do not mean to say that they ought never to be done, but I think they are usually adopted to overcome the nervousness of patients to anything like an "operation." Many a patient with hæmorrhoids, if he is told that he is not going to be operated upon in any way, but the pile injected, will submit to it. A man will submit himself to injection of his pile with carbolic acid who would not consent to have it ligatured or crushed. Therefore it may be desirable to carry out such treatment where the patient is too foolish or too nervous to have a better operation done. Such individuals would gladly submit to electric currents, the use of the X rays, or anything that combines mystery and inutility,—facts well known by irregular practitioners in the surgery of the rectum. With regard to operations by clamp and cautery, and crushing,

Digitized by Google

they have this disadvantage—that when done, even by the best surgeons, they are sometimes followed by hæmorrhage. And when I mention these complications to you I want to tell you that they are what I have personally seen. With regard to the operation for crushing piles, it is said by those who advocate it that crushing the base of the pile does away largely with the after pain. There is a great deal of truth in this. The crushing does seem to materially modify pain. But the sloughs separate, and occasionally hæmorrhages occur. I have seen this operation done in this hospital largely years ago, by the late Mr. Pollock, and though the results were excellent in most cases, trouble with hæmorrhage occasionally occurred. The operation I advise as the one generally to be done for internal piles is the somewhat old-fashioned ligature operation. I was once performing this operation and a surgeon of another hospital was present, and he said to me, "I see you still carry out the surgery of Hippocrates!" I have no great opinion of the surgery of the Ancients, generally speaking, but there are a few old operations which I believe are better than some modern ones, and ligature of piles, properly done, is one of them. I may also point out that this is the operation generally selected by those London surgeons who are the most engaged in rectal surgery at the present time.

Before operating, the patient needs careful preparation. If he be an alcoholic and over-fed man you will carefully prepare him for a week previous to operation by cutting off his alcohol, giving him blue pill and saline draughts, and getting rid in that way of the engorgement of his portal system. If you operate upon an alcoholic at once with no preparation, he is very apt to get delirium tremens —a very awkward complication. You go to visit your patient after a pile operation to find him dressed and sitting up, laughing, and talking nonsense about walking out or going to his club! I have seen such an occurrence, and have had to order male nurses to restrain the patient by force. This is not a pleasant occurrence after a bad pile operation. Another most important preparatory detail is to empty the colon of all fæcal material. If you do not empty the large bowel some hard masses of fæces will descend after the operation and press upon the sore, tender parts. This will give the patient great pain, and you will have much trouble in getting the mass away. Therefore, not only empty the rectum before you operate, but the colon also. And the best way to bring this about is to give the patient the night before a full dose of castor oil—half an ounce to one ounce; one ounce if he is a strong, plethoric individual. Doctors seem to be very much afraid of these old-fashioned purgatives, and they think that two or three spoonfuls of oil is enough. Let the nurse, on the morning of the operation, give two or more copious enemata, so that the whole of the descending colon and rectum is absolutely cleared of fæcal matter. The patient may be placed in one of two positions. Some surgeons prefer them on the right side with the legs and buttocks drawn up. Others, like myself, prefer the lithotomy position. You dilate the sphincter—and this is very important for the patient—to avoid that troublesome painful spasm which otherwise occurs. The forefingers or thumbs are passed into the anus and pressure exerted in various directions, both transversely and vertically. The stretching is continued for three or four minutes until the sphincter is felt to be quite relaxed and flaccid. I have observed operators dilate the sphincters in so forcible a manner one would think they were going to rend the patient asunder. Stretching should be firmly and gradually done in all directions, taking some time over the performance, so that on conclusion the anus remains patulous. Then the piles show themselves, and they can be readily seized by a proper instrument. I think the best instrument is not forceps, but Salmon's hook, which was used by the inventor of this operation, and which you will see in instrumental collections. The pile is drawn down, and next the nerves leading to it are cut by successive snips with blunt-pointed scissors at the junction of mucous membrane and skin on the lower or anal aspect. Do not include any skin in the part to be removed. The after cicatrisation is remarkable, and you must allow for it at the time of operation if you wish to avoid contraction after. This cut at the base of the hæmorrhoid can be made tolerably deep. This is a little practical hint of great importance, as avoiding after pain in the operation. By a deep cut you divide the cutaneous nerves, and do not tie them. The pile is left attached by mucous membrane and by some vessels. It is a practical fact of great importance that the main vessels of internal piles run from the

Digitized by Google

superior aspect, and not from the lower. If you commit the error of snipping the pile all round you are apt to cut a small vessel which may bleed into the bowel above your ligature. Next apply the ligature. The material should be thick soft silk, not hard corded silk such as is often used. The silk should be well waxed, and the "waxing" should be done with beeswax and iodoform. If the silk be waxed thoroughly, it makes all the difference with regard to being able to tie it very tightly and thoroughly strangulating the tissues. The ligature should be tied as tightly as you can. Here it is easy to commit another error. You do not thoroughly strangulate the pile. It will not come away, and in about a fortnight's time the patient may have to again take an anæsthetic to get the operation completed.

Now comes the very important point as to what to do with the hæmorrhoid after it is tied. The majority of modern operators, I think, cut the piles off. You can do this if you like, but you expose yourself to a risk in so doing. The ligature may slip over the little stump, and after-bleeding occurs. So I advise you to stick to the old-fashioned plan of the inventors of this operation. I was familiar with the practice of some of the leading authorities on this subject in my student days, and they invariably returned the strangulated piles into the bowel. Their reason was that it was then quite impossible for any slipping of the ligature to take place. If you cut off the pile close to the "stump" you run a risk of the slipping of the ligature in the night, and the consequent very troublesome hæmorrhage. The strangulated piles, then, are returned within the sphincter. The next part of the operation, and a very important part, is to pack a little iodoform wool within the patulous sphincter, and put a soft tube into the rectum. My common practice is to place this tube in the centre of the pad, and this prevents the troublesome distension of the wound with flatus. The patient is placed back in bed with a firm perineal pad and bandage. A small suppository of morphia and belladonna is introduced. The first complication you will have to deal with afterwards is retention of urine. This may last for several days. It is to be met by passing a soft catheter into the bladder, preceded by the injection into the urethra of a 2 per cent. solution of cocaine. Be very careful that your catheter is aseptic. Many nervous patients look upon the passage of a catheter as an operation of itself, and they will dread it being done. Therefore it is better to tie a soft catheter in such cases into the bladder at the time of operation, and then the patient is saved further trouble. Every two or three hours the nurse bathes the anus with hot (1 in 40) carbolic acid lotion.

The great trouble of the patient after the operation—and, indeed, to him, the principal part of the case—is his first action of the bowels. The patient will begin about the second or third day to say, "This is all very well, the pain is better, but I shall never be able to allow my bowels to act." No one can adequately realise the terrible soreness about the anus after an operation of this kind, and what happens when the bowels act. Unless you are careful you get into great trouble with such a case, because if you give a mild purgative you will find the nervous patient will so fear the action of the bowels that he will retain his motion and the purgative also. So it has been my practice to give early on the morning of the fourth day two calomel and colocynth pills and a saline draught to follow, or a full dose, one ounce of castor oil, followed in two hours by a large cup of warm tea. But I think it is well always to get the trouble over in the *daytime*, and not at night. Here is a point of importance. What position is the patient to be in for the early actions of the bowels after the operation for piles? If you permit him, as is, I fear, a common practice, to sit upon a pan, or seat, or night commode, you run the risk of hæmorrhage. It has long been my practice for the first ten days to always make a patient pass his bowel evacuations lying upon his side, with a piece of mackintosh and a pad of tow under him. Thus straining and congestion is largely obviated. In very nervous patients I find it a most useful thing, just before the bowels act, to throw an enema into the rectum, consisting of two or three ounces of sweet oil and a little laudanum. This may be injected with a No. 12 catheter without hurting the patient. And, more than that, I always, immediately before the bowels act, instruct the nurse to brush over the margin of the anus with 3 per cent. solution of cocaine. With all these aids the patient has what is practically a not very painful evacuation. The anus is thoroughly bathed with hot carbolic lotion after each evacuation. These details may seem

Digitized by Google

very trivial to you, but when you get a nervous, sensitive patient, you will find that he will appreciate these little points more than you can express. It is upon these sort of details that a surgeon especially founds his reputation of getting his patients through troublesome operations without pain. As the case progresses the patient must have light diet, and every two or three days his bowels must be opened by castor oil and an oil enema. The pain and soreness gradually subside, and about the eighth or tenth day the ligatures come away, and after that you allow the patient to get up on the sofa. Remember the next all-important point in the after treatment of internal hæmorrhoids. Keep the motions soft by confections of sulphur, or senna, or cascara, for about six weeks after the operation. If you allow patients to have a hard motion they will tear open the newly healed wounds.

Another very important point is to occasionally, a fortnight after the operation, gently pass your finger, smeared with white precipitate ointment, into the contracted sphincter. This dilates the part and prevents contraction, and the ointment facilitates the healing of the wounds left by the removal of the internal hæmorrhoids. Supervision should be established until the wounds have thoroughly healed. This is a sketch of the way to conduct the operation for piles by ligature. It is old-fashioned, and often brought into discredit by a surgeon not doing it properly. Piles are missed and left, or they are not sufficiently strangulated, or the ligatures are allowed to slip off. Too much tissue is removed, and after contraction occurs. Ligature of internal piles is not easy to do, and it requires great care to carry it out properly.

Regarding the troublesome complication of secondary hæmorrhage, many years have elapsed since I have seen such an accident after operation for piles. It is generally due to the practice of snipping off the piles too close to the ligatured stump. I have already alluded to this. When the ligatured stump retracts into the bowel the tissues relax, and what seemed a very tight knot outside becomes a very slack one inside. You will say, why not transfix the pile with a ligature? This is a practice which is very prevalent, and many excellent surgeons do it. My objection is that you should never transfix a vein with a ligature if you can help it, because of the possibility of setting up septic thrombosis. If you are unfortunate enough to get secondary hæmorrhage after a "pile operation," the kind of symptoms which you encounter are these:— The patient, on the night or day of the operation, or after the first action of the bowels, will say he has an urgent desire to evacuate; and out comes a large quantity of clot into the tow or bed-pan, looking like a mass of liver. And the nurse gets seriously alarmed, especially when in ten minutes or so he says, "I must go again." And again out comes a quantity of more blood. The patient gets pale in consequence of the loss of blood, and terror, and you make an examination. Your finger passes into a rectum which is dilated with masses of blood-clot. I trust none of you will ever be brought into contact with such a troublesome case as this; but you must look the enemy "in the face." It is of no use to put an ice-bag on the abdomen and "hope for the best." You have to again give the patient an anæsthetic, again dilate the sphincter, put on a forehead light, or have strong illumination of some sort. Sponge the part out and search for the bleeding point. Having found it, you may be able to pass a curved needle beneath and tie it with silk. Or you may be fortunate enough to be able to catch the source of bleeding in a pair of pressure forceps, and leave them hanging out of the anus for two or three days. But if there is a general oozing, and you cannot stop it or tell where the bleeding comes from, you must plug the rectum. I think the best instrument for this purpose is Benton's tampon bag. This is an india-rubber bag with a central tube, which is put in a collapsed state up the rectum and then blown out. The central tube allows the gas to pass through, and the pressure of the india-rubber stops the bleeding. It is something like the tampon for stopping epistaxis. I think this is better than plugging with sponge. But if this accident occurs in an out-of-the-way place, and you cannot procure the bag I mention, one of the best ways of stopping hæmorrhage is by taking a sponge cut into the shape of a wedge or bell, putting a rectal tube through the centre of it. Grasp the bell-shaped sponge with a pair of strong forceps, and pass it up the rectum. When the forceps are withdrawn the sponge expands and exerts pressure upon the bleeding points, the tube allowing gas to pass through. The sponge is gradually removed by cutting it away piecemeal with scissors after-

Digitized by Google

wards. In any form of rectal plugging you must be careful of the central tube, or you will cause a species of intestinal obstruction, which adds to the trouble already existing in no small degree.

I think these are most of the leading points regarding the treatment of piles. And once more before we part, recollect the immense importance of making a general examination of the patient as well as a local, and pay attention to the general medical as well as to the surgical treatment. It is one of the greatest errors to suppose that because a patient walks into your consulting room with piles he is to be at once operated upon. You must examine him thoroughly, and you must by means of proper drugs and dietary prepare him for any operative treatment which it is right to carry out. No cases are more often badly managed than these. They are thought "trivial" or "minor" by most students and many surgeons. I fear the "spectacular" element still enters largely into the reasons why most students attend operations; and hence the common operations of surgery are relegated to a short study in a text-book, accompanied by the solace of a pipe. Think what bad practice it would be to at once operate upon piles in a man who was suffering from cirrhosis or cancer of the liver. Again, think of the bad practice of operating for piles upon a man who is also suffering from a cancer high up in the rectum. You will thus see that it is necessary to think a great deal about a case of rectal disease, and carefully think upon the symptoms of each case; otherwise you will make a failure of it, and run the risk of falling into grave disrepute.

Cavendish Place, W.;
February 13th.

New Post-syphilitic Stigma.—Nobl found the sign he describes in 18 per cent. of 150 syphilitic subjects free from any perceptible manifestations of their disease; that is, in thirty-seven subjects whose infection dated from two to twenty years before. The sign to which he refers is the appearance of the scrotum, indicating atrophy and involution after infectious infiltration. He describes the appearance as an arrangement of the wrinkles on the surface in the form of circles and arcs, causing patches of a delicate tracery. The patches are a bluish grey in colour, and are evidently the result of regressive metamorphosis, and may prove useful in establishing a retrospective diagnosis.—*Journ. A. M. A.*, January 23rd.

APPENDICECTOMY.

A discussion at the meeting of the Hunterian Society, held at the London Institution by

Mr. Harold L. Barnard .	The London Hospital.
Mr. George Lenthal Cheatle	King's College Hospital.
Mr. James Berry . .	The Royal Free Hospital.
Mr. James Sherren . .	The London Hospital.
Dr. W. H. B. Brook . .	Lincoln Hospital.
Mr. Hugh Rigby . . .	The London Hospital.
Mr. Michels	The German Hospital.
Dr. W. H. Best . . .	Ilford.
Mr. Cuthbert Wallace . .	St. Thomas' Hospital.

Dr. F. J. SMITH in the Chair.

(*Concluded from p. 272.*)

Mr. JAMES BERRY remarked that in the main he agreed with the rules that had been laid down by Mr. Barnard. With regard to the avoidance of ventral hernia, he thought it important, if possible, not to cut any of the large nerves supplying the rectus muscle. If a large incision were made in the abdominal wall it was very likely that one or more of those nerves would be cut, and that would lead to paralysis and atrophy of the muscular fibres, and so favour the occurrence of ventral hernia. He did not think it mattered much whether in the interim operation catgut or silk were used, provided that the silk were extremely fine. Personally he generally used No. o silk, and he had never seen any trouble from its use in this operation. He did not employ coarse silk.

Referring to the removal of the appendix during an acute attack, he thought that the risks of the operation were far more serious when performed at that period of the disease. During the acute attack the septic focus was not limited to the interior of the appendix, as it was in the interim period. It was at first diffused over a more or less considerable area of the peritoneal cavity around the appendix. Appendicectomy at this stage undoubtedly involved risk of disseminating that septic matter very widely. In the great majority of cases of acute appendicitis this septic matter was, in the course of a few hours, localised by the exudation thrown out by the peritoneum. Removal of the appendix might be performed with considerable safety in the first few hours after the onset of an attack, but subsequently there was considerable risk of disseminating this septic matter and setting up general sepsis. As to the

Digitized by Google

question *when* the operation should be done, he should condemn all those people whom he might call the extremists. There were some men who said, "I do not approve of removing the appendix at all. I leave the case, and if an abscess forms I do not open it until it points." This was a view which he personally had never upheld and thought to be entirely wrong. Nevertheless he thought there was no doubt that the mortality in appendix cases which were not operated upon at all was much less than most people thought. But to leave an appendix abscess unopened for an indefinite period was to run an unnecessary risk. Then there were extremists in the other direction, people who would remove the appendix in every case of appendicitis, acute or otherwise, directly they saw the patient. That might be good practice if the case were seen within the first twelve hours of the attack, but they knew very well that hospital surgeons did not often see cases at this early period. They saw them most often on the second, third or fourth day after the attack had commenced, and he thought that as a general rule removal of the appendix in those cases was not advisable. The results, as far as these could be obtained from hospital returns, bore that out. He had searched pretty extensively four or five of the surgical registers of various large London hospitals for statistics on this point, and he was appalled by the mortality in connection with the hospital cases of removal of the appendix during the acute attack. There were various published statistics pointing in the same direction as those quoted by Mr. Barnard; in one instance it was shown that out of forty-nine cases of acute appendicitis operated upon on the third, fourth or fifth day as many as twenty-one died.

He pointed out the difficulty of laying down definite rules for the treatment of acute appendicitis. It seemed to him that harm was very often done by operating for acute appendicitis. He thought they must all have seen cases operated upon in the acute stage where death had obviously been the direct result of the operation, whereas similar cases treated in olden days without operation had got perfectly well. Twenty years ago, when he was house-surgeon and house-physician, cases of appendicitis were common enough in the wards. Operations in the acute stage were rarely performed, and deaths very seldom occurred.

With regard to the question of interim appendicectomy, he certainly agreed with Mr. Barnard that it showed a very small mortality. It was a simple operation, attended with scarcely any risk, and extremely satisfactory in every way. And he did not see why they should necessarily wait for a second attack. There were, no doubt, some cases where the patients were able to take care of themselves and rest and lie up during their attacks, and in such cases there was not the same necessity for removal of the appendix as in a good many hospital cases. He thought that as a rule it was well to advise the patient to have the operation done after the first attack, and to explain to him what risk he ran if he put it off and did not have it done.

The subject of *appendicular colic* was an extremely important one. He thought that these cases were eminently suitable for operation. The pain of true appendicular colic was due to the tension of the distended appendix, strictly comparable to the pain of biliary or renal colic, which was now known to be due to over-distension of the gall-bladder and renal pelvis respectively. He summed up his views on the question under consideration by saying that on the whole he agreed that appendicectomy might be done in many cases within the first twelve hours of a first attack of appendicitis. In all ultra-acute cases immediate operation was urgently demanded. After twenty-four hours he did not think removal of the appendix should be performed save in quite exceptional cases. In subsequent attacks he did not think that in the majority of cases appendicectomy was at all necessary in the first twenty-four hours. In most abscess cases he would certainly not remove the appendix. In the opening of an appendix abscess the greatest care should be taken not to foul the unaffected portion of the peritoneal cavity. Cases of appendix abscess seldom required incision before the fifth day, although there were undoubtedly many exceptions to this rule.

Mr. SHERREN said he quite agreed with what Mr. Barnard had said with regard to the method of performance of appendicectomy in order to avoid ventral hernia and sepsis. He quite agreed that the appendix should be removed after the first attack of appendicitis and after appendicular colic. *Appendicular colic* was due to increased tension

Digitized by Google

within the appendix unaccompanied by local peritonitis, a condition that was not often seen; he could not remember seeing more than three or four such cases. He was very much struck with one case under the care of Mr. Barnard—a girl of about eighteen years of age, who had sudden attacks of pain in the appendix region associated with marked hyperalgesia in the "appendix triangle;" these attacks came on about every six or eight weeks. The appendix was removed, and was found packed full of concretions; there was no peritonitis round the appendix. He remembered two other cases in which there was stricture and no signs of peritonitis; both these cases showed well-marked hyperalgesia. He felt quite sure that in these cases the appendix ought to be removed. There were several points on which he did not quite agree with Mr. Barnard. He could not agree to the removal of the appendix in all cases of appendicitis within the first twenty-four hours of the first attack, for even were it possible to see all cases during this period, there were many cases that could wait until the quiet interval for removal of the appendix, and gain the benefit of the extremely small death-rate for removal at this period. He thought the ideal treatment was to watch the cases through and remove the appendix in the quiet interval, but unfortunately this was not always possible. There were cases in which the appendix must be removed within the first twenty-four hours, and these were cases coming under observation with signs of perforation into the general peritoneal cavity. If one is able to see a case within the first twenty-four hours, and well-marked hyperalgesia is present in the usual situation, there is certainly no need to operate and remove the appendix; but if no hyperalgesia is present, or if present disappears, then the case was certainly one for appendicectomy.

The appendix often perforates locally or into a cavity shut off by adhesions, or more rarely into the general peritoneal cavity during the days subsequent to the first attack, so that he could not agree with Mr. Barnard's direct negative that the appendix should not be removed during the subsequent days of an attack of appendicitis. The high mortality in the cases operated upon during the third, fourth and fifth days was due to the fact that many of them were cases of general peritonitis—cases which had come under the notice of the surgeon too late, and which should have been operated upon on the first or second day. Supposing that one were watching a case in which hyperæsthesia was present, and on the second or third day this hyperæsthesia disappeared without improvement in the general condition, the temperature perhaps remaining the same, but the pulse-rate increasing, operation should be performed, the appendix removed, as disappearance of hyperalgesia without improvement in the general condition of the patient is due to perforation or gangrene of the appendix. Abscesses in most cases are due to perforation of the appendix later than in the first twenty-four hours. By operation on the disappearance of the hyperalgesia, if we do not save the patient from peritonitis we save him from an abscess, and of course from the risk of a subsequent attack of appendicitis. A large number of cases will go through these days without needing appendicectomy. The cases in which the appendix should not be removed during these days were those which were obviously getting well, and include a large number of all the cases of appendicitis. If the patient came under observation on the third, fourth or fifth day with well-marked hyperæsthesia, the temperature high, and the pulse-rate high, then operation would be necessary, and in all probability an abscess would be found, and on removal of the appendix it would be found unperforated. Then again there were cases within that period where the patient was ill—the temperature might be 101°, the pulse rapid, and there was no hyperalgesia present. These cases should be operated upon and the appendix removed; it would be found gangrenous locally or generally. In a case of that sort the patient would be saved from peritonitis or from the development of an abscess.

In case of appendix abscess he (Mr. Sherren) thought that careful search should be made for the appendix. In most cases, from the position of the abscess, one was able to make a good guess as to where the appendix was. With a good light and careful sponging out of the cavity, he thought that the appendix could be removed in most cases without adding to the mortality. In cases of general peritonitis he had nothing further to add to what the previous speaker had said.

Dr. W. H. B. Brook said he had come there

Digitized by Google

with the hope of learning something on a question which was of such great importance to general practitioners. The difficulty that they found in the country was the question as to whether a case should be operated upon in the first twenty-four hours of the first attack, or whether it should be left. During the last few days he had looked up the cases that he had seen during the last ten or eleven years, and he could not find a single case, occuring in his own practice, that had died during a first attack of appendicitis. An important point was how to distinguish between appendicitis and appendicular colic. That was the question which they so often had to decide in the acute cases. As a rule, when a case was seen for the first time complaining of pain in the region of the appendix, with a certain amount of temperature and possibly some sickness, one had found that if the patient was treated locally and medicinally, the attack generally in the course of a few days passed off. In every case sent to him for the first time complaining of pain in that region, he would be rather loth to advise the patient immediately to have the appendix removed. He was extremely interested in what Mr. Sherren said about appendicular colic, because he had two cases under his own care of patients who had had two or three attacks of appendicular colic. He removed the appendix, and in neither of those cases was there any sign of peritonitis or perforation having occurred; in each the appendix was shaped like a flask with a narrow neck about one-eighth of an inch in diameter, and at the end it was distended until it was quite the size of one's little finger. Both of these cases had had repeated attacks without any peritonitis, but simply with pain, and in each of them the appendix was removed perfectly, easily, and with great satisfaction to the patient, who had no further recurrence. He should like to know whether there was any one who had had any experience of the value of making a leucocyte count in first attacks of appendicitis. What they had to distinguish between, it seemed to him, were the cases that could be tided over the first attack and the cases that fulminated and would kill the patient invariably within a couple or three days if nothing was done. He always made a point of watching the pulse every two or three hours, and latterly he had in several cases made a leucocyte determination at a short interval, and if he found the leucocytes increasing or the pulse running up steadily, increasing by five beats every hour, he would advise an immediate operation. He would like to hear what Mr. Barnard had to say on that point. He was always on the look-out for rigors as showing the formation of pus; and whether a case in which there were no rigors might be left was also a point of importance.

Mr. HUGH RIGBY expressed his agreement with Mr. Barnard in the main, but there were certain points in which he differed from him entirely. In the first place, he considered that Mr. Barnard made a mistake in limiting the dangers of appendicectomy to "the formation of ventral hernia" and "sepsis." Many other difficulties might arise during this operation, in comparison with which the above complications mentioned were of somewhat minor importance. He alluded to the possibility of not being able to actually find the appendix at all, and the extreme difficulty and risk incurred in removing an appendix firmly adherent to the intestine or pelvic viscera.

As to the technique of operation, he thought that splitting of the muscles of the abdominal wall was perhaps the best method, but that by incising the rectus sheath, pushing back the rectus and opening the peritoneum underneath it, a much better means of getting at the appendix and almost as good after-results could be obtained. With regard to the suturing material, he agreed with Mr. Berry that silk for ligaturing the appendix and its mesentery answered quite as well as catgut, especially if the silk were very fine. He could not see the object of using Nos. 2, 3, or 4 silk in appendicectomy. As to the sewing up of the abdominal wall, he used buried silkworm gut for that purpose. The silkworm gut might be boiled for a quarter or half an hour before use, and thereby made sterile. If the muscles of the abdominal wall were split, he hardly thought that buried sutures were at all necessary.

Concerning the question of when to operate, he agreed with Mr. Barnard in most points. At the hospitals the trouble was that cases came in on the third, fourth or fifth day after the onset of the attack. One hardly ever saw them during the first twenty-four hours. He quite agreed with a previous speaker that they should never, if

Digitized by Google

possible, touch a case during the third, fourth or fifth day, but that in some cases an immediate operation was unavoidable.

One point about abscess cases; *i. e.* on the eighth, ninth or tenth day Mr. Barnard advocated opening the abscess, but making no attempt to feel or remove the appendix. He (Mr. Rigby) thought they always ought to put their finger into the abscess cavity—not necessarily to feel for the appendix only, but to feel for concretions and sloughs and to remove these, the formation of a sinus after abscess depending in many cases on the presence of concretions.

In cases of general peritonitis due to a sloughing appendix he could not see the object of operating without attempting to remove the appendix. If one merely put in tubes one left the chief cause of the mischief behind. He would certainly try to get at and remove the cause.

Mr. MICHELS supported the remarks of the last speaker with regard to the mode of operating. He went on to remark that appendicectomy was not always a simple and easy operation. He remembered having had cases which were amongst the most difficult he had had in abdominal surgery, and where there were many adhesions to the small intestine and cæcum, and it was with great difficulty that he got the appendix away. He would recommend in very difficult cases the proposal of a colleague of his in Germany, simply to slit open the appendix, to excise the mucous membrane from its interior, and to close it again. That was a less dangerous operation than removal of the appendix at any price. It had happened to himself twice, he was sorry to say, among fifty or sixty cases, that he could not find the appendix. Both were cases in which there had been a great number of attacks, and the appendix was either destroyed or buried in adhesion. He closed the abdomen again and watched the cases, and they both did well. Of course it was a great disappointment to him, but it had done no harm to the patients.

Then about appendicular colic. One sometimes removed the appendix in these cases, and on examining it found practically nothing of a pathological character in it. Those cases invariably were greatly benefited by the operation. It was, he thought, very strange that among his cases of that kind the great majority occurred in women.

With regard to the question of operating within the first twenty-four hours, was it always possible to make a diagnosis within that time? One case he particularly remembered in private practice on which, for some reason, he did not operate, but felt sure that it was a case of appendicitis, and the case turned out to be one of typhoid fever. In a case of peritonitis he would make it a point to remove the appendix as a source of infection whenever possible. He had had six cases of general peritonitis within the last three years, and had had the pleasure of seeing four of them saved.

Dr. BEST said he had been very much interested, in talking to general practitioners, to notice how variable the experience of different men was in general practice. Within the last fourteen or fifteen years he had had certainly a rather unusual number, for a general practitioner, of cases of appendicitis. He had been led to think that the apparent exaggeration of some surgeons with regard to the fatality of unoperated cases was not really exaggeration. His experience was that a very fair proportion of strictly acute cases of appendicitis died, and he had unfortunately seen a very fair number, especially within the last ten years, where the friends of the patient would not agree to an operation, and the patient had died before it could be performed. In talking especially to country practitioners he noticed that many of them had a very small number of cases of appendicitis. Previous to coming to London he himself was in the country two or three years, and there he had only very few cases. He was now in a rather large and rapidly growing London suburb, and he had an unusual number of cases of the kind—as many as two in one week. He would only add that anything which tended to make the general practitioner think that delay was at all excusable was likely to do a lot of harm.

Mr. CUTHBERT WALLACE remarked, with regard to the incision to be adopted, that McBurney's incision was, he thought, a good one. He was brought up on it, and had seen it used a great deal. The only objection to it was that if the appendix was adherent near the pelvic brim the surgeon was placed in rather a difficulty, even if he had two strong men to retract for him; otherwise he thought it was a most excellent incision.

Digitized by Google

An incision through the rectus sheath was, he considered, also a good one; it could be extended if necessary and sewn up very easily, and the results, as far as he had been able to judge, were very excellent.

As to the mortality in cases of localised abscess, he thought it was occasionally due to the fact that the presence of a general infection of the peritoneum had been overlooked and the abscess simply opened. This was an argument for a bolder policy or the use of an incision on the left side to ascertain the condition of the peritoneum. He did not say that one would have saved such cases if one had opened the abdomen and washed it out. In looking up the cases in St. Thomas's Hospital which had occurred since the year 1895, he had come across three in which the abscess was opened and the patients died within twenty-four hours, general peritonitis being found at the post-mortem.

With regard to the time for removing the appendix, he thought that most observers had come to the conclusion that the interval after the first attack was the right time. That course had been adopted for some time at his own hospital. He might raise the question of the recurrence of abscess. A man was certainly not safe, even though the appendix had suppurated and an abscess formed and the wound healed up. The other day a boy died within a year after an abscess had been opened, from a second attack of appendicitis and peritonitis.

He thought that operation within the first twenty-four hours was the ideal thing. Americans adopted that method quite early, but now they were swinging round to removing the appendix in the quiescent interval. Removal of the appendix on the third, fourth or fifth day was no doubt attended by a most awful mortality. That was due to the fact that by that time the patients had become very ill, and were consequently sent to the hospitals, and the surgeon operated on a diffuse peritonitis and rightly removed the infectious source, namely, the appendix. If left without interference these cases die all the same, but the mortality would not be put down to the appendicectomy. In the early stages the question of operation and removal of the appendix must of course usually depend on whether the case was improving or getting worse. In cases of general peritonitis Mr. Barnard seemed to think that it was hardly any use doing anything more for the patient. In conclusion, Mr. Wallace drew attention to the fact that if the appendix was left *in situ* there might be left untouched what was practically a great hole in the cæcum if the organ had sloughed at the base.

Mr. BARNARD, in reply to these various criticisms, remarked that Mr. Rigby thought ventral hernia and sepsis dangers "of somewhat minor importance" when compared with failure to find the appendix and adhesions to pelvic viscera. Mr. Rigby seemed to be in some confusion as to what were the dangers in appendicectomy and which the difficulties. Whilst there was no doubt that a surgeon in difficulties was a danger to his patient, he could not see that failure to find the appendix at all was a danger unless the surgeon unduly prolonged his search for it; nor could he see how the adhesion of the appendix to the ovary or tube was likely to lead to a worse result; and as to adhesions to gut, if they led to ill-effects at all, it was by sepsis.

The splitting operation by McBurney had been criticised by Mr. Michels, Mr. Rigby, and Mr. Wallace, who all appeared to prefer the vertical incision through the outer part of the rectus sheath. He freely admitted that this incision was much more convenient for a pelvic appendix, and he always employed it when there was reason to believe the appendix was there; but it was just as inconvenient for an external appendix running up towards the liver, and, further, it had the great disadvantage pointed out by Mr. Berry that if it was extended far up into the abdomen, several nerves were divided and the lower segments of the right rectus muscle paralysed. Another objection to this incision was that it divided the anterior sheath of the rectus, and this structure below the umbilicus transmitted the transverse strain of the abdominal muscles, and its division without proper subsequent suture was liable to lead to separation and consequent ventral hernia.

The material to be buried in appendicectomy was a very important matter. He agreed that very fine silk was innocuous, but for the stump of the appendix itself he preferred catgut. He could not agree with Mr. Rigby that silkworm gut was a good material to bury in muscle. The sharp wire-like

Digitized by Google

ends produced constant pricking, and if they reached the skin led to sepsis. The objections to silkworm gut were the same as to silver wire, and very few surgeons used that now.

But there were surgeons who used No. 4 silk for ligaturing the stump of the appendix. Why they thought it required that strength of silk he failed to understand. Only the other day a patient remarked that she supposed it was the usual thing to wait a year or so for the suture to come out of the wound, because Mr. So-and-So had operated on her sister, and when the ligature came away it was stout silk.

A most interesting point had been brought out by the country experiences of Dr. Brook and Dr. Best and the comparison the latter was able to make with his subsequent town experience. It appeared clear that appendicitis was not only rarer in the country, but that it was of a much milder character. That in unoperated cases deaths hardly ever occurred. He was glad, however, to find that Dr. Best's town experience went to show that in cities it was a very serious malady, and one requiring the vigourous measures he (Mr. Barnard) had recommended.

As to the removal of the appendix during the quiescent stage, there seemed an unanimity of opinion that it should be done after the first attack where possible, and even after appendicular colic where this could be diagnosed with certainty and a competent surgeon were available.

Mr. Barnard said he differed from Mr. Berry's ideas about colics. He thought that colics were not so much due to distension as to tension. Whatever produced tension, cramp or spasm of plain muscle-fibres produced colic. Distension of the urinary bladder, the gall-bladder, or the pelvis of the kidney might, by stretching the walls of these cavities, lead to peristaltic contractions, and so to colic, but even in these colics he believed the chief pain was due to the bile-duct or ureter grasping the stone and straining to pass it on. He had seen typical biliary colic due to the passage of a gall-stone along the cystic and common duct after cholecystotomy when a tube was in the gall-bladder, and in the same way he had seen renal colic from the passage of a clot or stone down the ureter when a tube was in the pelvis of the kidney. In these cases there could be no distension. On the other hand, colic might occur without any distension. How, on his distension theory, did Mr. Berry explain the strangury of the empty acutely inflamed bladder and the intestinal colic due to lead and calomel?

As to the question of operation during the first twenty-four hours of the first attack, there seemed a fair agreement that where possible and where the symptoms were acute the appendix should be removed; but on the question of operation during the second, third, fourth and fifth day there was great disagreement, and still more so as to whether the appendix should be searched for and removed from every abscess cavity.

Mr. Cheatle, Mr. Sherren, and Mr. Wallace advocated removal of the appendix in practically every abscess, and Mr. Cheatle appeared to think that the abscess wall, far from being a beneficent structure, was positively harmful. He (Mr. Barnard) had seen many cases in which pus had been let through into the general peritoneal cavity as a result of the surgeon's interference with that abscess wall. Mr. Cheatle's point about rapidly acquired immunity was a most interesting and important one, but he, in Mr. Barnard's opinion, was blinded by it to the value of the abscess wall. If he were prepared to tear down the adhesions after he had evacuated the pus from an abscess in order to remove the appendix, and pointed triumphantly to the fact that spreading cellulitis did not start from his wound as a proof of this all-powerful immunity, he ought logically to be prepared to sew up his wound after this procedure, and leave the patient to deal with what little pus was left by his immunity. The fact that he left his wound open and drained it showed the great advantage he really attached to all measures—of which the abscess wall was certainly the most powerful—which diverted this pus from the deep uninfected parts and directed it out of the body. Indeed, if Mr. Cheatle wanted to understand the use and function of the abscess wall, he must remember that in all domains of life noxious bodies were extruded in a similar way.

Mr. Barnard agreed that he had not alluded to the point raised by Mr. Rigby that the abscess cavity should be carefully searched for concretions or sloughing appendices loose in it, a method which he thoroughly endorsed. However, he could not admit, as the same speaker had said, that there was no object in operating in general peritonitis without removing the inflamed appendix. He (Mr. Barnard) regarded the appendix as a bomb. It was very important to remove it before it really exploded, but when this had occurred and the building was wrecked, the fragments of the bomb might be safely left to be swept away with the rest of the *débris*, and required no special treatment or attention.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 591. WEDNESDAY, FEBRUARY 24, 1904. Vol. XXIII. No. 19.

CONTENTS.

PAGE

*Pain: Its Importance in Diagnosis and its Tendency to Mislead. By ANTHONY BOWLBY, C.M.G., F.R.C.S. ... 289

*Hyperæsthesia of the Stomach. By W. SOLTAU FENWICK, M.D., M.R.C.P. ... 296

*The Treatment of Tuberculous Disease of the Tarsus. By LAWRIE MCGAVIN, F.R.C.S. 300

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London E.C.*

PAIN:

ITS IMPORTANCE IN DIAGNOSIS AND ITS TENDENCY TO MISLEAD.*

By ANTHONY BOWLBY, C.M.G., F.R.C.S.,

Surgeon to St. Bartholomew's Hospital.

GENTLEMEN,—The relief of pain is justly held to be one of the chief duties of the medical profession, and the causation of it is often a problem upon the correct solution of which depends the ability to afford relief.

I do not therefore propose to consider this evening the actual means at our disposal for the relief of pain, but rather its value as a symptom and its importance in diagnosis.

In the first place I would ask you to consider very briefly the utility of pain, although to those who suffer it this utility must ever appear of little value. But looked at in its much wider application, it must be remembered that the sense of pain is essential to the preservation of every tissue. Consider for a moment what is the common fate of structures that are insensitive. When the eyeball is deprived of its ability to perceive the presence of foreign bodies no measures are adopted for their removal, and ulceration and destruction of the globe are common consequences. If the nerves of the hand are severed, the fingers are often burnt or cut and otherwise injured, because such lesions cause no pain, and whitlows, ulcers, and even gangrene may result. Similarly if the spinal cord be divided, the distension of the bladder causes no pain, and severe cystitis may ensue from over-distension, while bedsores in such patients are mainly due to the same want of sensation in parts exposed to pressure and contamination by urine. And what is true of the external parts of the body is true also of the deeper parts and the viscera. The

* An address delivered before the Hunterian Society of London.

Digitized by Google

pain that follows fracture of a bone or injury to a joint enforces that rest which is essential to repair. And the pain of inflammation of the pleura or the peritoneum similarly enforces the rest which is necessary in order to prevent the spread of the inflammation. In the viscera also when the sense of pain does not give that warning of danger which suggests care, there is incurred a much greater risk to the patient, so that the man with typhoid fever who has no sufficient pain to keep him in bed, eats his ordinary food and goes about to the detriment of his chance of recovery.

Pain must therefore be regarded as a means whereby all sentient beings are kept constantly informed of that which is hurtful to them. It is designed to preserve all the tissues from the effects of injury which might otherwise pass unnoticed or uncared for, and to this extent it is beneficial. To this extent alone, unfortunately. For beyond this power to give a friendly warning, pain is liable to become one of our worst enemies. But I do not propose to occupy your time in discussing this latter aspect of my subject. For my purpose is rather to impress upon your attention the need of giving very careful consideration to those complaints of pain that may be utilised in arriving at a diagnosis, and to point out how pain that may be misleading if it is not understood may be of the greatest utility when properly appreciated.

At the very threshold of such an inquiry we are met at once by the fact that pain is in most respects more a subjective symptom than one that can be justly measured or estimated by any surgeon. We are, indeed, to a very great extent dependent on the statements of patients as to the pain they suffer. For not only may such statements be much exaggerated or even intentionally misleading, but it must be admitted that the perception of pain varies very greatly amongst different individuals, and sometimes to such an extent as to make all ordinary experience misleading rather than informing. Yet amidst much which is misleading there are nevertheless some rules to guide us in estimating the importance of pain, and I will endeavour to place such before you. In the first place I should feel inclined to say that a rule that has no exceptions is, that complaints of severe or persistent pain in all very young children are invariably genuine. And we have, further, in young children none of that patient endurance of pain and none of its concealment which make it so difficult to estimate its intensity in many adults. I am therefore very strongly convinced that pain takes the most important place amongst the symptoms of disease in early life and very seldom leads entirely astray. And I am consequently always inclined to put out of court at once the many specious names often given to complaints of pain. For I believe that in the use of such names as "growing pains" and "rheumatism" there is often the obscuration of important symptoms, and in many such cases the real disease is never sought for, even perhaps never mentioned to a medical attendant. Consider the countless cases of tubercle of bone and joints where, in the early and the curable stage, the inability to use the limb freely, the disinclination for active exercise are all passed over and neglected, and how often even the next complaint of more definite and serious pain is also put aside with the suggestion that it is "rheumatic." Such cases as these will show you what I have in my mind when I say that severe or persistent pain in a child always has a definite cause and is never misleading if due attention is given to its investigation. I often wish, indeed, that the very name "rheumatism" did not exist, for it seems to be considered a sufficient explanation of almost every ill, ranging from disease of the brain or spinal cord to hip disease, acute osteitis, or psoas abscess. Of one thing I am sure, that if a child is said to suffer from obscure rheumatic pains, it is tolerably certain that its troubles have nothing whatever to do with rheumatism, and that the sooner the case is definitely investigated, the better chance there will be of curing the patient.

And at the other extreme of life also, there are pitfalls to avoid. In many of the diseases of old age there is not the same intensity of pain as in earlier years, and consequently there are not a few cases where one is misled by the absence of the usual characteristic symptoms. Look, for example, at the intense pain caused in a child by a tiny vesical calculus and compare it with the tolerance of the bladder in many old people which results in the stone causing only the slighest discomfort. I have twice seen old men who complained of a little bladder irritation without any pain at all, and from whom I removed, in the one case five large calculi, and in the other six. Yet one of these patients was leading an active life in the country, and came

Digitized by Google

to town because he thought he had a stricture. Compare again the pain of a strangulated hernia in the case of a child or young healthy adult and in the case of an old man. In many of the latter the man denies all pain, and I have on more than one occasion even known patients to be walking about with gangrenous intestine in the sac. But if we are not prepared for such possibilities and are guided alone by the characteristic symptoms seen in young and vigorous subjects, we should sadly be astray in our diagnosis and treatment.

And next I would ask you to consider the very different accounts that different patients will give of pain when their conditions are quite similar. I once saw a man whose eyeball had suppurated after corneal ulceration of six months' duration. He said that he thought it began "by something getting into his eye while he was cutting a hedge;" but he had not been to a doctor, and had gone on with his work as a farm labourer as if nothing was the matter. He was anæsthetised for the removal of the globe, and then under the upper lid was found a large thorn, which presumably had been there for six months. It is difficult to conceive how the man could have tolerated such conditions for even a few days without making some attempt to obtain help. But he said that his eye did not hurt him much, so he did not trouble about it.

This, of course, is an extreme case, but it shows how obscure may be the perceptions in people of dull intellect, and there are not a few patients of this class from whom we can obtain no assistance in diagnosis by any complaint of definite pain.

On the other hand, there are patients whose perceptions are so keen and whose nervous system is so highly strung that they respond to the slightest stimulus, and complain of pains out of all proportion to the troubles that cause them. I am not alluding for the moment to patients whose pains are wholly imaginary, but rather to those who exaggerate and feel excessively a pain that is real. Such patients are often people who have a keen appreciation of many other matters besides pain, and amongst them are often found those who are talented as musicians, artists, or scholars, etc. Their appreciation of pain, indeed, is only on a par with their other perceptive faculties. They are the very reverse of the dull, stolid, ignorant labourer, to whose mind all art would appeal in vain, and who is similarly apathetic to pain also. We have to learn to recognise these over-sentient patients, however, for unless we appreciate their temperaments we shall certainly be misled by them when they are really trying to explain as clearly as they can the sensations they perceive and the pain they endure. And it must be remembered that such people as these are able to give a much more graphic and impressive account of the pain than is the ordinary patient. They are consequently liable to be all the more convincing in the impression they leave upon their hearers. Experience alone can enable us to judge of the intensity of pain in such cases, but in order to do so it is necessary to appreciate the difficulties I have alluded to.

More troublesome still are the patients in whom not only does no cause of pain exist, but in whom there is no real pain at all. These are mostly the people who are classed either as neurasthenic or hysterical subjects, but some of them of course are mere malingerers. I shall not pretend to deal with these at any length, but there are one or two points to which I should like briefly to allude. In the first place it must be remembered that all the people who suffer from neurasthenia and the allied conditions are not either females or of an emotional disposition. The neurasthenic state can be produced in men who are naturally robust and vigorous by accident, illness, excessive mental strain, alcoholism, or indulgence in drugs. I have seen it produced by all these, and more often by a combination of two or more. Thus I have known pain and partial paralysis follow in one case where a mere contusion was caused by a fall from a horse. And in another of a bullet-wound across the shoulder muscles which did not touch the spine. In many cases of prolonged illness and suffering the normal resistance of a healthy nervous system becomes overtaxed and broken down, so that at last a strong man may be reduced to a pitiable condition of constant querulous complaining and inability to bear the slightest pain or discomfort. Nothing, I think, however, has so rapid an effect as the administration of morphia. It was a very common and proper practice in South Africa to administer morphia to the wounded men, in order to facilitate their transport in ox wagons and other vehicles. But unfortunately in many cases the relief thus obtained suggested the continuance of the drug in hospital, and very likely later on on board ship,

Digitized by Google

with the result that the patients were reduced to such a condition of hysteria or nervous breakdown that they became quite unable to control their feelings, and complained and even cried over pain that really no longer existed. No more typical cases of neurasthenia could be seen; but fortunately the remedy was comparatively simple. For the stopping of the morphia supply soon brought about the cessation of the symptoms.

I would also like to say a word of warning as to the rather frequently accepted view that in hysterical women who complain of persistent pain the threat or the actual performance of an operation is likely to do good. It may perhaps have that effect on those who are merely shamming, but it certainly does not have it on many others. I have known a woman succeed in having her abdomen opened by three different surgeons for pains that were purely neurotic in origin, and I have known others submit to more mutilating operations, which they have persuaded surgeons to perform in the belief that there was some real cause of pain that could be removed. Quite the most remarkable case of this kind, however, that I am myself acquainted with is one I will relate, in order to show you to what extent these patients will submit to operation. A young woman of about thirty was admitted to St. Bartholomew's Hospital under the care of Mr. Alfred Willett a few years ago. She had suffered, it was alleged, from a condition of elephantiasis of the left thigh, and for this amputation had been performed near the hip-joint. She now complained of a similar condition of the other lower extremity. She said the swelling had been increasing for a year or more and caused her intense pain and discomfort, for the relief of which she wished to have a second operation performed and part with her remaining leg. She was apparently in constant and extreme pain, and was very insistent that, as the other operation had relieved her, a second amputation should be performed at once. An examination of the limb showed a condition of so-called "spurious elephantiasis" of a very extreme kind. The whole limb was immensely swollen and misshapen, the leg was shapeless, and the ankle was hidden in the general swelling. The thigh and knee were similarly altered, and the whole limb was heavy and useless, so that the patient was quite unable to walk and the knee could hardly be bent on account of the general rigidity. The tissues were very firm and did not pit on pressure, the skin was thick and rather livid; but there were no evident enlarged veins on the one hand, and no dilated lymphatics on the other. The most noticeable thing about the extent of the swelling was that it did not fade away gradually, but was very abrupt and limited by a sharp depression close to the trunk. And it was this abrupt line of demarcation that suggested the remote possibility of the whole condition being artificially produced, unlikely though it seemed. Yet such was the case, for one night the sister of the ward suddenly turned the bedclothes down and revealed a handkerchief twisted like a tourniquet round the upper part of the thigh, and it became evident that the case was one of so-called hysterical malingering. I have described this patient's condition because I think that it is very instructive. Not only is it remarkable that such a condition as elephantiasis can be produced by artificial means, but it is still more extraordinary that a young woman who was certainly not otherwise insane should actually have parted with one of her limbs for a condition wilfully induced by herself, and was also most anxious to induce a surgeon to remove the remaining leg. After such a case as this we may well be cautious in expecting that operations will prove of benefit for the relief of hysterical ailments, and it only remains to add that under massage and exercise the so-called elephantiasis in this patient was completely cured and the limb gradually restored to its normal condition.

Thus far have I dealt mainly with the appreciation and perception of pain by patients of different ages and temperaments, and I wish now to turn to another aspect of my subject, namely, the localisation of disease as indicated by pain, and the difficulties that result from pain being referred to localities at a distance from its site of origin—what has been often called "referred pain."

It is of course perfectly well recognised that in certain parts the pain due to disease is often referred to a distant region which is both more sensitive and also more often the seat of sensations derived from contact with external surroundings than is the affected area. So that the pain in the knee in hip disease, at the end of the penis in a case of vesical calculus, and that in the testis in a case of renal calculus are commonly estimated at

Digitized by Google

their true value, and the real seat of disease is not overlooked. But although such commonly known conditions as these are fully appreciated, I think that there are many others which are very insufficiently appreciated, and to some of them I propose to refer, as well as the time at my disposal will permit. In the first place let me point out that in many affections of the spinal cord, patients at any rate, and practitioners sometimes, are easily put upon a false track by the referred pains which are so common in tabes and other forms of sclerosis. It is the commonest possible thing for these pains in the lower extremities to be called "rheumatic" or sometimes "neuralgic," and in very many cases the patient makes his own diagnosis of what he calls rheumatism, and in not a few instances imposes his diagnosis on his doctor, who falls into the trap solely because he does not examine the patient with the idea of some spinal-cord trouble in his mind. The troubles of micturition that so often complicate tabes are similarly put down to cystitis or enlarged prostate solely because the idea of tabes is not entertained.

There is, however, one diagnosis due to "referred pain" of which I am even more suspicious than I am of "rheumatism," namely, "sciatica." What numberless diseases have been lumped together under that misleading title, and how extraordinary it is to see over and over again that so long as a pain can be given a name that is commonly recognised the patient seems to be perfectly satisfied. Let me illustrate my meaning by referring briefly to a few illustrative cases. A civilian living in India began to suffer pain in one thigh, for which he consulted a surgeon. They agreed between them that it was due to "sciatica," but no examination of the part was made. Medicine was tried and then various local remedies, though without avail. But after suffering a good deal for several months other advice was sought. The surgeon who now saw the patient examined him carefully and soon found a deep-seated swelling in the pelvis on one side, and the man afterwards came to England, and was under my care until he died of a large sarcomatous tumour of the ilium.

A woman of about forty went into a hospital for "sciatica," for which she had been treated for several months, and while walking in the ward her femur spontaneously fractured near to its neck as the result of a malignant tumour, which had destroyed the bone in this region.

A young lady had suffered for several months from persistent "sciatica," for which many remedies had been tried without avail, and in order that I might suggest some other treatment I was consulted. The diagnosis was considered to be so settled that an examination was deemed rather superfluous. But when I did examine the gluteal region I found a large abscess, which was due to tubercular disease of the adjacent bone.

I could easily multiply such examples, but I have said enough for my present purpose. I wish to remind you that tubercular abscesses and tumours may affect the sciatic nerve, and may cause a pain which is the pain of sciatica, but I also want to remind you that almost all these conditions, when overlooked, are overlooked solely because a diagnosis is made without an adequate examination of the affected parts and of the pelvic region. Few of these conditions would be missed if carefully looked for, but I should like to make an exception of the pain due to those carcinomata of bones which may result from the dissemination of a cancer of the breast or other parts, for it is quite extraordinary how extensively the affected bone may be destroyed without any tumour being perceptible, and, indeed, in many cases without any tumour that causes a swelling being present; for there may be no real enlargement of the bone. I saw a lady some years ago who was supposed to have sciatica, and whose breast I had removed some time before for cancer. She said that she had suffered from "sciatica" for some months, and the back of the thigh was so sensitive that I examined her under an anæsthetic. I could feel nothing, although she was very thin, but I strongly suspected a cancerous growth of the bone, which I could not perceive, and a short time later this opinion was only too fully confirmed by the occurrence of a spontaneous fracture of the femur. I have seen other similar cases, and it is to be noted that in all of them there was, in addition to the pain, a sensation of weakness as if the limb would give way, and this sensation really conveys a proper appreciation of the actual condition of the bone.

I would now ask you to turn your attention to another region of the body and another class of case altogether. Yet a class in which referred pain is a very important symptom whose true import it

Digitized by Google

is not always easy to appreciate. The abdomen is the part I allude to, and the many viscera it contains are all of them liable to cause reference of pain to regions at a distance from the seat of disease. But I do not propose to deal seriatim with all the viscera, and shall confine myself chiefly to the intestines and the peritoneum.

I think it may be laid down as a general rule that pains originating inside the intestine are not felt at the place where the affected coil lies unless the peritoneum is involved, and that even then, although there may be local pain, that the pain is largely referred to the region of the umbilicus, and is also felt more or less over the front of the abdomen. It is never referred to the back and loins as are the pains that originate in the stomach, the kidneys, the liver, and the uterus, with the exception that the rectum when affected often causes aching pain in the sacral region. I think that the consideration of cases that are commonly met with will confirm this statement, for pain at the umbilicus is commonly complained of in the various kinds of colic, where the peritoneum is not involved, as well as in most cases of strangulated hernia, where the peritoneal coat is definitely affected.

But there is one part of the intestinal tract to which I wish to allude more especially, namely, the appendix. It might well be thought that too much has been written lately of this very minute portion of the bowel, yet as far as the question of referred pain is concerned I think there is yet room for further comment. I think it may be granted in general terms that it is accepted that appendicitis causes pain in the right lower half of the abdomen, and that in the absence of pain in this region, appendicitis is not usually suspected. I have, however, been much struck by the number of cases in which this localising pain is entirely absent in very many attacks, and how in patients who suffer from relapsing catarrhal appendicitis it often happens that several of the earlier attacks pass away without any local pain in the iliac fossa being complained of, and without appendicitis being suspected. In these cases the history is one of attacks of pain more or less severe or protracted, coming on without obvious cause, associated with some tympanites, sickness, or retching, and with a varying amount of pyrexia, but with no particular pain in the appendix region. In these patients there is rather a generalised abdominal pain, felt especially in the region of the umbilicus. Here are a few notes of some such cases to illustrate the point I wish to emphasize, and to indicate how this referred pain may prevent the surgeon from even suspecting the real cause of the patient's troubles.

A young lady of twenty-two gave the history that for two years past she had been liable to suffer from very severe attacks of abdominal pain. The attacks came on suddenly without any obvious cause, and were in no way associated with the taking of food, with constipation, or with the occurrence of the menstrual period. They caused intense cramping pain with nausea and flatulence, the pain being most acute on the left side of the abdomen and above the umbilicus. The attacks at first occurred at an interval of two or three months, but during the past year had become much more frequent as well as more severe. They sometimes caused such collapse that of late morphia had been administered, and the patient had gradually given up all active habits and had begun to lead an invalid life. She had been under the care of a most capable practitioner, and had seen very many consultants, especially with reference to the possible causation of the pain by some ovarian or uterine disease, and gastric ulcer and gall-stones had also been suggested. An examination showed that the patient was a slightly built but quite healthy girl, very anxious to be cured, but rather neurotic from all she had gone through. The character of the pain, apart from its locality, was such as to suggest that after all it might well be due to the appendix, and when an attack was subsiding I found on examination that she admitted to some slight tenderness in the right iliac fossa, although this was so overshadowed by the pain in the left and upper part of the abdomen that she was herself unaware of any tenderness there. There was certainly no tenderness elsewhere in the abdomen, and the patient readily submitted to operation on the understanding that it might be merely of an exploratory nature. I found, however, a much thickened and very long and bent appendix, with slight adhesions, and containing fæcal material. There was no affection of the generative organs, and no other discoverable abdominal disease. Since the date of the operation nearly a year ago there has never been any return of the old severe pain, and the patient has returned to her usual habits. For some time she had slight abdominal discomfort, but

Digitized by Google

this appears now to have almost entirely passed away.

In the next case the patient was a healthy young man who for about eighteen months had been liable to attacks of severe abdominal pain with sickness and fever. No cause had been found, and the pains were localised at and around the umbilicus, there being no pain in the appendix region. After twelve such attacks, however, it was noted by a physician who had seen him on several previous occasions that on the next occasion there was pain in the right iliac fossa after the acute general pain had subsided, and I now saw the patient in consultation. The physician told me that in all the other attacks the pain had been so completely misleading that he had no thought of the appendix, but that now he had begun to think that all the other attacks had really been due to appendicitis. I agreed with him and operated, finding an extremely thickened and inflamed appendix fixed by recent adhesions to the neighbouring mesentery.

In a third case the patient was a young boy who had attacks of so-called "stomach-ache," for which he had frequently been laid up several days at a time. They were supposed to be bilious attacks, but no definite cause could be discovered. The pains were epigastric, were accompanied by vomiting and distension, and had become so frequent as to cause great anxiety. I saw the boy at the end of one of these attacks, and found that he had definite tenderness near the cæcum, although this had not previously been noticed, and I subsequently removed a much diseased appendix, with the result that the patient was completely relieved from further pains.

I have within the past two months operated on two patients at St. Bartholomew's Hospital, in each of whom there was a history of several attacks of general and severe abdominal pain without any history of either pain or tenderness in the right iliac fossa, and in each patient a much diseased appendix was found and removed.

I will not trouble you with further histories of such cases, but in a dozen or more patients I have been told similar stories of absence of all localising pains in any of their attacks of appendicitis, and with the reference of all symptoms to the centre or even the upper part of the abdomen.

It must be admitted at once that such cases are necessarily puzzling, but they are not nearly so likely to be misunderstood if it is fully recognised that there are really many cases of appendicitis in which the pain complained of is felt all over the abdomen. I say advisedly the "pain complained of," for it will be found on examination that in many of these patients as the attack subsides the pain is felt in the right iliac fossa when it has subsided elsewhere. But, just as in cases of severe renal colic, the lumbar pain may be completely masked by the general abdominal pain, so that, unless inquired for, all characteristic renal pain is unnoticed, so here also the severe pain referred to the umbilical region will so completely mask any local pain that the right iliac fossa is not complained of. And just as, again, in renal colic the renal symptoms may become evident when the general pain subsides, in these cases also when the attack is passing away both pain and tenderness may be discovered in the appendix region when, if not looked for, they would pass completely unnoticed and uncomplained of.

These cases may be diagnosed also to some extent by a process of exclusion. What causes are there for such attacks as I have described when all stomach and ovarian troubles have been eliminated? What else except a moveable kidney or some band of adhesion which occasionally entangles a coil of intestine can cause in a perfectly healthy patient recurrent, sudden, and severe attacks of abdominal pain, which are nevertheless transient, and may leave the patient none the worse for their occurrence? Such a history alone is suggestive of appendicitis, but it is to be noted that it is often only the earlier and slighter attacks that occur without localising pain and cause merely general abdominal pain, and that in later attacks the characteristic local symptoms begin to assert themselves.

The explanation of this is not, I think, difficult. I believe that in all these cases when the pain is general it will be found that the appendix is free in the peritoneal cavity, being adherent, if at all, by its distal portion alone to the mesentery or intestines, and that when it becomes inflamed it causes merely the general pain of a reddened and inflamed peritoneum, and so a reference of pain to the umbilicus and the whole peritoneal cavity. When the appendix inflammation spreads to the cellular tissue of the iliac fossa or of the mesentery,

Digitized by Google

or when it causes adhesions in the iliac fossa or to the psosas muscle, then the pain will be felt largely in the iliac region, whether or no it be referred to the general peritoneum as well. And it may be laid down as a general rule that the more the pain is purely local the more likely is the inflammation to be limited to the immediate neighbourhood of the cæcum. The absence of local pain in the earlier and slighter attacks is thus easily explained, for in many cases of catarrhal appendicitis there is for a long time no extension of acute inflammation to the neighbouring peritoneum and consequently no adhesions, but as the attacks recur and become more intense the cellular tissue of the mesentery or of the iliac fossa becomes involved, adhesions form, and the pain becomes more localised.

Very much more might well be written of the referred pains of the other abdominal viscera, but I feel that I have said quite enough for my present purpose.

I have tried to bring before you the necessity of nquiring very fully into the histories of pain as given us by our patients and of realising the difficulties of appreciating its intensity in different people.

I am certain that very many errors in diagnosis arise from not sufficiently investigating the exact characters and situations of pain, and from accepting too easily the diagnosis of the patient as to the cause of this suffering.

The subject of "referred pain" alone is well worthy of your continued study and thought, for in very many instances a referred pain traced to its original cause supplies the key to an otherwise difficult diagnosis.

Manchester Square, W.;
February 18th.

X-Ray Treatment of Uterine Cancer.—The results which may be expected are wonderful. The vacuum tubes bring about complete relief from pain, and the entire treatment results in a cessation of the discharge, disappearance of the odour, which in these cases is often most offensive, and a restoration to general health. Fibroids diminish somewhat in size, and the progress of the carcinomatous infiltration is arrested. Each treatment acts as a general tonic, and the patients often declare that they would be treated every day if they could afford it.—*Therapeutic Gaz*, vol. xxviii, No. 1.

HYPERÆSTHESIA OF THE STOMACH.*

By W. SOLTAU FENWICK, M.D., M.R.C.P.,
Physician to Temperance Hospital and to Evelina Hospital for Sick Children.

GENTLEMEN,—An exalted sensibility of the mucous membrane of the stomach probably occurs in many diseases of that viscus, both functional and organic, and tends considerably to aggravate the subjective sensations which ensue from these varied disturbances of digestion. As a primary disorder, however, gastric hyperæsthesia has received very little attention, and the symptoms to which it gives rise are usually attributed either to inflammation or to ulceration of the stomach. The frequency with which it is observed in medical practice, and the difficulties which seem to be encountered in its recognition and appropriate treatment may serve as an excuse for my remarks upon the clinical features of the disease.

Hyperæsthesia of the stomach sometimes appears during the course of such diseases of the central nervous system as cerebral tumour, meningitis, tabes dorsalis, neurasthenia, and hysteria; and in the last-named complaint an access of gastric pain and vomiting is sometimes the immediate precursor of a convulsive seizure. In women prolonged lactation, menorrhagia, excessive leucorrhœa, or some other debilitating condition often appears to excite it, while in men sexual excesses, masturbation, mental overstrain, continued deprivation of food, and even the abuse of tobacco may exert an equally deleterious effect. Occasionally direct irritation of the gastric nerves by stimulating foods, alcohol, coffee, ice, or the use of certain drugs like quinine, digitalis, salicylate of sodium, or iodide of potassium is responsible for an attack. A sudden return to a full diet after a long abstention from solid food is often followed by the symptoms of gastric hyperæsthesia, and this fact probably explains the epigastric pain and vomiting which are sometimes observed during convalescence from enteric fever. In rare instances the administration of chloroform provokes an attack which may persist for several days. But of all the conditions which

* Abstract of a lecture delivered at the London Temperance Hospital.

Digitized by Google

predispose to or excite the complaint, profound anæmia is infinitely the most common in this country, and consequently the majority of the patients who come under medical observation are young women between the ages of sixteen and twenty-five, who for some time have suffered from increasing pallor of the lips, dyspnœa on exertion, and other indications of chlorosis. In out-patient practice at this hospital the disease constitutes about 12 per cent. of all cases of dyspepsia.

Under normal circumstances the processes of digestion are not accompanied by any subjective symptoms, and consequently the healthy individual is quite unconscious of his possession of a stomach; but if from any cause the secretory or motorial functions of the viscus become deranged, his attention is at once attracted to his digestive organs by the pain, distension, and flatulence which ensue from stagnation and fermentation of the food. In like manner the development of undue sensibility of the mucous membrane of the stomach engenders a sensation of discomfort or pain whenever particles of food are brought into contact with the tender tissues, while in the more severe cases the degree of irritation may be so severe as to produce vomiting. It therefore follows that pain and vomiting are the two most prominent symptoms of gastric hyperæsthesia.

If a case be watched throughout its course it is found to present four stages of development, each of which is characterised by some feature of clinical importance.

The first or initial stage is accompanied by symptoms which for the most part are common to several other forms of dyspepsia. In the early morning the patient experiences giddiness or faintness upon rising from bed, accompanied by nausea, flatulence, headache, or palpitation. The appetite is rarely impaired, but immediately after each meal fulness and discomfort are felt in the epigastrium and left hypochondrium, which culminate in an aching or burning sensation that tends to radiate over the adjacent parts of the thorax. Gaseous eructations occur from time to time, and are apt to be followed by nausea and palpitation. The bowels are usually confined, but occasionally a lienteric form of diarrhœa ensues after the first meal of the day. The patient is markedly anæmic and low-spirited; she is also irritable and capricious in her tastes, and usually suffers either from amenorrhœa or from irregular catamenia. The tongue is pale and flabby, the urine copious and sometimes phosphatic, and frequent perspirations are apt to occur after the slightest exercise or excitement.

The second stage of the complaint is characterised by the substitution of veritable pain for the previous discomfort. It may now be observed that as soon as food is swallowed there is a sudden access of a burning pain in the region of the stomach, which continues with varying severity for one or two hours, and is accompanied by distension, flatulence, nausea, and palpitation. These symptoms are induced by every form of nourishment, and although most severe after indulgence in solid food are readily excited by hot and cold liquids, and even by milk. The bowels are now almost invariably confined, the appetite is diminished, and intense thirst at night is a common cause of complaint.

After a variable period the occurrence of vomiting ushers in the third stage. At first the attacks of emesis are only occasional and merely produce a partial evacuation of the gastric contents; but the tendency to sickness rapidly increases until it occurs after each meal, and leads to the rejection of the greater part of the ingesta. This form of emesis presents three somewhat peculiar features. In the first place, unlike that which occurs in cases of gastric ulcer, the rejection of the food does not exert any notable effect upon the pain, and even when the stomach has been completely evacuated the painful sensations only subside in a very gradual manner—in other words, the surface of the stomach continues to ache after the exciting cause has been removed. Secondly, even after the vomiting has continued for several weeks the patient fails to exhibit any obvious signs of emaciation, and if she is weighed regularly the loss of weight appears to be trifling when compared with the severity of the gastric symptoms. Thirdly, unlike most other diseases in which vomiting is of frequent occurrence, the appetite continues surprisingly good, and the patient will often continue a meal after she has vomited the first portion of it. At this period the anæmia makes rapid progress, constant headache is often present, and the catamenia are either very scanty or suppressed.

The fourth stage is characterised by failure of the general nutrition and by an alteration of the symptoms, which, if the case only comes under observa-

Digitized by Google

tion now for the first time, is apt to present considerable difficulties of diagnosis. So rapidly does vomiting follow the ingestion of food that the pain which was previously experienced disappears, and the epigastric discomfort complained of between meals is usually attributed to the retching and straining. In not a few instances streaks of blood are vomited from time to time, and attacks of syncope occur, which create great anxiety and alarm. The bowels are obstinately confined, loss of flesh is rapid, the anæmia is profound, and a sense of internal weakness obliges the patient to remain in bed. The ease with which the disease is cured by medical treatment renders it comparatively rare for it to terminate in a fatal manner, but occasionally death ensues from exhaustion, syncope, or from some intercurrent affection of the lung.

Physical signs.—Tenderness is invariably present, and affects the whole region of the abdomen occupied by the stomach. It is usually more marked in the left hypochondrium over the site of the fundus than in the epigastrium. Cutaneous hyperæsthesia over the same area exists in about two-thirds of the cases, and may also occasionally be detected beneath the left breast and below the inferior angle of the left scapula. The localised tenderness which exists in the epigastrium in cases of gastric ulcer is invariably absent. The stomach itself rarely presents any signs of dilatation, and if the vomiting is excessive the viscus usually appears smaller than normal. The gastric contents obtained after a test meal vary in different cases, being normal in character in about 60 per cent., somewhat deficient in hydrochloric acid in 10 per cent., and exhibiting moderate hyperchlorhydria in 30 per cent. Lactic acid is always absent, and the motorial functions of the organ are normal. Occasionally scybalous masses may be felt in the transverse and descending portions of the colon. Examination of the blood shows a great diminution of hæmoglobin, the percentage amount of which may fall as low as 20 in severe cases, and rarely exceeds 40.

Diagnosis.—There are two diseases with which gastric hyperæsthesia is frequently confounded, both of which are unduly frequent in young and anæmic women, namely gastric ulcer and a painful neurosis of the colon.

In *gastric ulcer* pain rarely occurs until after the completion of a meal, and unless the ulcer is situated close to the œsophageal opening never ensues immediately a mouthful of food has been swallowed. It is chiefly produced by the direct irritation of solids, and unlike the pain of the functional disorder is at once relieved by a milk diet. The pain is referred to the epigastrium, which presents a small area of localised tenderness, while the rest of the gastric region is free from pain upon pressure. Cutaneous hyperæsthesia is rare. Vomiting is much less frequent than in gastric hyperæsthesia, is only provoked by food, occurs at the crisis of a painful attack, and by ridding the stomach of its irritant contents at once relieves the pain. Hæmatemesis occurs in the majority of the cases and is copious in amount. Rest in bed and a milk diet rapidly relieve the symptoms of ulcer, but produce little effect in cases of hyperæsthesia. The gastric contents in cases of chronic ulcer usually exhibit hyperchlorhydria.

A painful contraction of the transverse colon excited by the introduction of food into the stomach is a very troublesome complaint in certain anæmic girls who are members of a tuberculous family or who have suffered in early life from megrim. The pain occurs immediately after food, is often very severe, and may be accompanied by vomiting. It may be noticed, however, that it is usually excited by solid food or by hot fluids, that instead of being localised to the gastric region it is referred to the whole of the upper part of the abdomen, and that careful inquiry will usually elicit the fact that it commences in the right hypochondrium and extends thence across the belly about the level of the navel. The pain is far more intense than that of gastric hyperæsthesia, is colicky in character, and the complaint is readily cured by the administration of suitable aperients and by the prohibition of fruits and green vegetables.

Chronic Gastritis, for which gastric hyperæsthesia is occasionally mistaken, is rare in young women unless they are the subjects of valvular disease of the heart, phthisis, chronic nephritis, or are addicted to alcohol. Actual pain after food is rarely complained of, but about an hour or more after meals a sensation of discomfort and fulness occurs, followed by flatulence, acidity, and nausea. Vomiting is frequent both in the early morning before breakfast and after meals, and in both cases relieves the unpleasant symptoms which were previously present. The ejecta contain an excess of mucus,

Digitized by Google

but are usually deficient in hydrochloric acid. The food is undigested, and various products of fermentation may be detected in the filtrate. The appetite is diminished, loss of flesh is a prominent symptom, and the bowels are irregular rather than confined. Saline purgatives, combined with careful regulation of the diet and a course of alkalies and bismuth will most readily relieve the complaint.

Treatment.—If any doubt exists as to whether a patient is suffering from ulcer or from hyperæsthesia it is wise to assume the presence of the more serious complaint, and to treat the case in accordance with this supposition. This line of practice is not only of value from the point of view of safety, but is in itself an important help to diagnosis, since it is found that the pain and vomiting due to ulcer invariably subside when the patient remains in bed and is confined to a milk diet, while in cases of hyperæsthesia the same symptoms are usually aggravated by such measures. The treatment of gastric hyperæsthesia may therefore be summed up in the statement that in its main particulars it is the exact reverse of that adopted for ulcer of the stomach.

If pain and vomiting are severe the patient should maintain a recumbent position for a few days, but confinement to bed for more than a week should be avoided. Local applications over the region of the stomach often help to relieve the symptoms, and accordingly a small blister may be applied to the skin of the epigastrium, or the part may be painted each night with the liniment of iodine.

Diet.—Inasmuch as the gastric pain is induced by fluids as well as by solid food it is advisable to prescribe soft foods rather than liquids, and as soon as the symptoms improve to permit an ordinary mixed diet. With this object lightly boiled or poached eggs, milk puddings, jellies, and clear soups are at first allowed, to be followed subsequently by white fish, chicken, game, rabbit, sweetbread, tripe, calf's head, with a little potato, cauliflower, or spinach. At a later period the patient resumes her ordinary diet.

The medicinal treatment of the complaint should be solely directed towards the cure of the anæmia and the correction of the constipation, gastric sedatives and stomachics being valueless and enemata often harmful. When constipation has existed for some time, and especially if the colon is loaded with fæces, half an ounce to an ounce of castor oil should be given each morning before breakfast; or if the patient cannot take the oil a full dose of the mistura alba may be substituted for it. Subsequently a daily action of the bowel should be secured by the administration of an iron and aloes pill night and morning after food, or of some other mild aperient each night, such as cascara and maltine, the confection of senna and sulphur or the compound liquorice powder. For the treatment of the anæmia nothing is of greater value than the ammonio-citrate of iron, ten to fifteen grains of which may be combined with a drachm of the solution of bismuth and ammonium citrate, and given three times a day after meals. These simple measures rarely, if ever, fail to produce an immediate improvement in all the aspects of the case, and should be continued until a cure seems to have been accomplished. It must be borne in mind, however, that relapses are apt to occur, and accordingly the treatment should be repeated if pain after food is again experienced.

Harley Street, W.;
February 18th.

Relation of Epilepsy to Chronic Gastro-intestinal Disease.—McCaskey gives the history of the case of a woman æt. 27 years. Seizures had been recurring about every three or four weeks for nine years. The patient was somewhat under the average power of mental and physical endurance. Examination revealed chronic gastritis and colitis, and suitable treatment was instituted, but there was no substantial improvement in either the gastro-intestinal disease or the epilepsy during the time that the patient was under observation, which was perhaps two or three months. The putrefactive processes of the intestinal tract seemed entirely beyond control by either diet or medication combined with suitable local treatment, and the patient finally passed from observation unimproved. A second case was that of a boy of fifteen years presenting a history of irregular nocturnal attacks of *grand mal*, closely associated with exacerbations of a chronic disease of the stomach. In this case an apparent cure of the epilepsy followed a comparatively brief treatment, and the patient was remaining well three years later.—*Medical Record*, No. 1734.

Digitized by Google

THE TREATMENT OF TUBERCULOUS DISEASE OF THE TARSUS.

By LAWRIE McGAVIN, F.R.C.S.Eng.,
Surgeon to the Seamen's Hospital Society; Assistant Surgeon to the Hospital for Women, Soho Square; Late Surgical Registrar of Guy's Hospital.

My remarks have no claim to originality or novelty; they are the outcome of the belief, repeatedly forced upon me by a long series of cases seen in the out-patient rooms and wards of hospitals in London, that the surgery of tuberculous disease of the tarsus has not kept pace with the rapid advance of the science in the treatment of other pathological conditions. In spite of much that has been written to the contrary, there are perhaps few pathological states in all the domain of surgery more disappointing to the surgeon, or more disheartening to the patient, than that of caries of the tarsal bones resulting from tuberculous disease.

This fact is, I think, amply evidenced by the number of cases one sees going steadily from bad to worse, in spite of months, often of years, of painstaking treatment, and eventually arriving, after repeated recurrence of the disease, at the final stage, as far as the limb is concerned, of amputation through the knee-joint or the leg.

The reason of this unsatisfactory state of affairs is, I believe, to be found in certain facts, some of them well recognised, and others whose existence has perhaps not been sufficiently taken into account, or whose importance has at least been underrated. These may be considered under four different heads, viz. (1) those depending on the peculiar nature of the disease; (2) those depending on the anatomical arrangement of the part; (3) those depending on the surgeon's method of treatment; and (4) those arising out of the patient's dread of operation.

The question of how far these points may be applied to patients of good position I would lay aside for the present, and consider the matter from the standpoint of the hospital surgeon, in view of the fact that the poor are the greatest sufferers, both in point of numbers and from the vital importance to them of the integrity of their limbs.

1. FACTS DEPENDING ON THE PECULIAR NATURE OF THE DISEASE.—Here we must remember that we are dealing with a morbid condition which is above all insidious; for "like a thief in the night" it creeps, unnoticed at first, almost unfelt, giving so little evidence of its presence that it is passed over by the patient as a sprain or a twist, till valuable time is lost and irreparable damage done. Again, it does not necessarily start at one particular spot, but often originates in several foci, all of which will be in various stages of progress by the time the disease comes under notice. It is, too, a disease well known in other parts of the body as being resistent, prone to recur on very slight provocation, liable to sudden outbursts of uncontrollable energy, and at times, in marked contrast to its usual lethargic state, involving the whole organism when least expected in a universal conflagration, by such fatal conditions as general tuberculosis, meningitis, etc.

Another point to be borne in mind is that the second stage in all tuberculous processes, viz. caseation, occurs at a much earlier date than is generally supposed, and will usually be found to have commenced before the patient comes under observation, a firm grip having thus been obtained by the disease. Again, the synovial membranes which lie in the immediate neighbourhood of bones affected by tuberculous disease, although possibly free from tubercle themselves, are yet in a state of inflammation which, being slight, renders them an easy prey to the bacillus. Hence it is seen that we are dealing with a condition which, from its pathological aspect alone, is likely to tax the best efforts of surgery, and which must therefore be taken early and dealt with unsparingly if benefit is to accrue to the patient.

2. FACTS DEPENDING ON THE ANATOMICAL ARRANGEMENT OF THE PART.—The following points, when considered carefully, seem to present an almost insuperable barrier to the complete eradication of the disease, and are among the chief factors which combine to render the task of cure one of the most unpromising:—The number and extent of the synovial sacs of the tarsus; the large mass of cancellous bone of which it is composed; the number of bones into which this mass is divided; the intricate manner in which the synovial sacs wrap round and enfold the bones; the comparatively poor blood-supply of the tarsus

Digitized by Google

and its ligamentous surroundings; and last, but not least, the close proximity of the sheaths of the peroneus longus and other tendons, renders the tarsus one of the most dangerously perfect harbours for the tubercle bacillus that exist in the human body.

Now, if the position of the foot in the body be considered, especially with regard to the children of the poorer classes, it is easily seen that an organ so dependent, so badly protected from damp, injury, and from sudden changes of temperature such as are met with in this country, is but ill-adapted to withstand the inroads of such a disease as tuberculosis. Add to these points the possible coincidence of valgus and varicose veins in unhealthy adolescents, and the chances of inducing healthy reparative processes is reduced to a minimum.

3. FACTS DEPENDING ON THE SURGEON'S METHOD OF TREATMENT.—First among these must be mentioned the natural desire of every honest surgeon to preserve intact so important a member as the foot; and this system of conservative surgery, however laudable in the majority of surgical operations, is yet in certain cases apt to be overdone and eventually to defeat the end in view. Of these the condition under consideration is undoubtedly a case in point. It has been said that "desperate diseases require desperate remedies," and although tuberculous disease of the tarsus can hardly be said to fall into this category, it is none the less a disease which in its more advanced stages calls for more energetic treatment than is generally accorded to it. What has this treatment been? It may conveniently be considered under the following headings:

(*a*) *Rest, complete and prolonged, in rigid splints, aided by careful and nutritious dieting and abundance of fresh air.*—Such treatment to be successful requires both time and money, of which few hospitals can devote sufficient of the first, and fewer patients are in possession of the second. Consequently a fair chance of recovery is seldom possible, for six months may be a long sojourn in a hospital or convalescent home, but it is all too short for the cure of this condition. For well-to-do patients it is everything if commenced in time, but for the poor it is impossible, and the disease proceeds unchecked; thus time is lost.

(*b*) *The above combined with the inunction of soft soap.*—Were it not that the name of Hoffa* is associated with this addition to the treatment, most of us would perhaps be inclined to ridicule its use. Hoffa, however, maintained that the alkalinity of the soap was capable of neutralising the lactic acid of the diseased tissues, and so tended to cure. The method has not met with much success; and, indeed, it is difficult to see how a caseating focus situated deeply in the tissues could be affected by so mild an agent.

(*c*) *The injection of drugs.*—Of these it may be said that their name is legion. Some years ago the use of iodoform-glycerine was strongly advocated by Mayo Robson;† the same year Peniéres,‡ of Toulouse, exhibited certain cases to the Academy of Medicine which he had treated with an alcoholic solution of euphorbia resin; and Eve reported the case of a patient treated by Koch's tuberculin (tuberculin R.); while Cipriani‖ submitted certain patients to the injection of chinosol in watery solution. Durante's method by the injection of iodine was recommended by Campanini,¶ who reported several cases; and in the following year (1898) Hahn, of Mainz,** suggested and practised the injection of formalin-glycerine. Of these cases some, it is true, were not those of tarsal caries; certain of them were cases of synovial tubercle; but as this form is even more within reach than when caries is present, it stands to reason that if but slight success has been attained in them by the method of injection, still less can be expected where the bone is involved. Although some measure of success has been stated to have been met with, it must be obvious to most surgeons that it has been of a very meagre order, since none of the methods mentioned have come into general use. Indeed, "mere laying open, and, still more, injection of sinuses where there is disease of the tarsus, is absolutely useless in most cases."††

(*d*) *Finsen's and Röntgen's discoveries.*—Con-

* 'Münchner medicinische Wochenschrift,' 1899.
† 'The Lancet,' 1897, vol. ii.
‡ Ibid.
§ Ibid.
‖ 'Allgemeine medicinische Central-Zeitung,' 1897.
¶ 'The Lancet,' 1898, vol. i.
** 'Centralblatt für Chirurgie,' 1899.
†† 'The Operations of Surgery,' Jacobson and Steward, 1902.

Digitized by Google

sidering the brilliant results which these have recently shown in the treatment of lupus and rodent ulcer, their complete failure, at least so far, to check the progress of tarsal tubercle has come as a disappointment. The question must, however, be considered as being still *sub judice*, and therefore must be left to the future.

(*e*) *The erosion of carious bone.*—I now come to the treatment which is perhaps the most favoured at the present time, and which unfortunately is too often applied without sufficient regard to its limitations. How far are we justified in resorting to erosion of bone in the tarsus?

If we consider the disease in two different stages—viz. that in which the healthy skin is as yet unbroken, but beneath which an abscess is in process of formation, dependent upon some quite superficial lesion; and that in which sinus formation is already present,—we arrive at two different conclusions, for the first is a condition which may be dealt with entirely aseptically, while the second is quite the reverse. In the first, erosion may be carried out very freely, sterilised decalcified bone being used to fill up what would otherwise be a dead space, the skin wound being closed in the usual manner. Here there is little fear of sepsis, and although dissemination may be caused, as Watson Cheyne* has pointed out, in certain cases, (*e.g.* that reported by Edington,† in which the erosion of a palmar abscess gave rise to a tuberculous choroiditis), its occurrence is sufficiently rare to justify its probability being ignored.

In the second stage the presence of septic micro-organisms must be reckoned with, and these, by the time the case comes under observation, may, and probably will have penetrated into the farthest recesses of the labyrinth of synovial tissue and bone which forms the tarsus. What, then, is to be gained by attempting the complete eradication of the disease by this method? I fear it has but little to recommend it. Firstly, the danger of a general diffusion of sepsis, in the presence of sinus formation, and a part so richly composed of cancellous tissue, is by no means an imaginary one. Secondly, granting that success attends our efforts, a consummation as devoutly to be desired as it is improbable, it can only be brought about at the cost of so much interference with important structures that the limb remains a scarred wreck; and, what is especially to be avoided, the patient henceforth carries about with him a part which is even more likely than before to fall a prey to the disease on the receipt of some trifling injury. Thirdly, the skin of tuberculous patients is often extremely thin and delicate, especially over the dorsum of the foot; it thus frequently happens that even when an erosion has been performed perfectly in accordance with the strictest asepsis, so little vitality remains in the integuments, that in a few days union of the lips of the wound is found to have failed, and the result is then equivalent to the spontaneous occurrence of a sinus. At the risk of being considered somewhat dogmatic, I would answer the question of limitation in erosion of bone as follows:

1. It *should be performed, but not persisted in*, where the disease is limited to the outer surface of the cuboid or the inner surface of the scaphoid bones, provided that the skin is healthy and the prospect of primary union good.

2. It *may be performed, but not persisted in*, when the cuneiform bones are involved, and the sinus, if such is present, is situated at a good distance from the dorsal line of incision for Syme's amputation.

3. It is *quite unjustifiable* (*a*) in disease of the os calcis or astragalus, even if no sinus is present, which has gone on to caseation or suppuration; (*b*) when there are sinuses in connection with scaphoid, cuboid, astragaloid, or calcaneal disease.

It is, I think, generally acknowledged that if the case eventually comes to amputation the stump should contain no part of the tarsus; for this reason Pirogoff's operation is rarely performed, Syme's method being that generally employed. Now that which applies to the tarsus applies equally well to the tissues which overlie it; and therefore, if amputation is to be performed, *it should be done before the heel-flap has been injured by sinus formation, or by the extension of the disease to it; and most certainly before any evidence of implication of the tendon sheaths is present.* I do not mean to imply that all cases of tuberculous caries in which the tissues are affected necessitate amputation at a higher level than the ankle, but I am convinced that many cases come to require such treatment eventually, when, by avoidable delay and well-meant

* 'Tuberculous Disease of the Bones and Joints,' p. 168.
† 'Lancet,' 1899.

Digitized by Google

but unfortunate temporising with the disease, the chances of giving the patient a useful Syme's stump have been lost.

It is probable that in our endeavours to root out the disease by erosion we but open up and infect fresh cancellous tissue previously uninvaded, or even inoculate synovial sacs and tendon sheaths, which may inadvertently be injured by the action of a sharp spoon passed deeply and vigorously applied. And here I may say that having recently examined thirteen cases, by dissection, in which the foot had been removed after repeated but ineffectual erosion in the vicinity of the medio-tarsal joint, I found in all but two the sheath of the peroneus longus invaded by tuberculous granulation tissue, quite localised, and in close relation to the original seat of the trouble. This may or may not have been the result of interference, but it at least suggests the possibility of harm being done where good was intended.

4. FACTS ARISING OUT OF THE PATIENT'S DREAD OF OPERATION.—The natural and ever-present horror of operation which haunts the minds of so many patients is, in the face of measures demanding the removal of any part of the body, and especially of such an important member as the foot, one of the chief obstacles to the treatment of these cases; and it is extremely difficult to persuade them that the little discharging sinuses, to whose prolonged presence they have almost become accustomed, represent a disease whose persistence and insidiousness is a constant menace to their lives as well as to their limbs. In many cases a very long period of delay will have to be faced before the patient makes up his mind to the inevitable, and in the meantime the following questions will probably be put to the surgeon:

(*a*) Is there no alternative treatment?

(*b*) Can the foot be replaced by an artificial limb?

(*c*) Will the operation guarantee a cure?

(*d*) Shall I be able to earn my living as well as before the operation?

And then, when all have been answered with perfect honesty, too often the operation is dreaded, and eventually put off to "wait and see if the foot does not soon get better." Let us remember that with regard to tuberculous disease no saying is truer than that "procrastination is the thief of time." It is not always the patient himself who is responsible for the delay, for it is too often the fault of parents and "friends," especially in the case of the children of the poor in hospital. None are so obstinate as the ignorant members of the working classes, and it is upon such that the decision as to operation so often, unfortunately, depends.

Now it is amputation, and that at a much earlier date than has hitherto been the custom, that I would urge for these cases; and my answer to those who would object to it on the ground of unscientific mutilation, is this. In the first place, no reliance can be placed on palliative treatment; secondly, the majority of these cases come eventually to amputation when it is *too late*; thirdly, it is infinitely better that the patient should have the best stump that surgery can give him—*i. e.* that provided by Syme's method, at little cost to tissue and small danger to life—than that he should lose that chance by delay and have to undergo a much more serious operation at a later date, in order to obtain what is at best but a poor exchange, a stump formed by a Teale's or a Stephen Smith's amputation, or by that at the tubercle of the tibia. Lastly, *faute-de-mieux*, it is undoubtedly efficacious. If, now, the questions enumerated be considered, it will be found that all of them except the first can be satisfactorily answered in the affirmative. Palliative treatment and erosion have been considered, and there remains only tarsectomy, partial or complete. Partial tarsectomy has little to recommend it, for in view of the fact that the extent of the disease is usually an unknown quantity, it is unscientific, and always leads to much deformity, and often to a perfectly useless foot; the cavity thus produced is, moreover, difficult to keep aseptic.

Complete tarsectomy, Mickulicz's osteoplastic resection, has been little practised in this country,* but would appear to have given some slight measure of success abroad, although Fenger, quoted by Treves,† mentions that out of thirteen cases done for caries of the tarsus, two died some six or eight months after the operation from general tuberculosis. This operation cannot, however, compare favourably with Syme's amputation, either in utility or in æsthetic appearance; indeed, were the patient to be shown a case, or a photograph of the

* Stiles reported a successful case in 1900.

† 'Manual of Operative Surgery,' 1897.

Digitized by Google

hideous deformity produced by this method, it is more than probable that he would at once choose amputation as being a less disfiguring operation. Further, it is not unlikely to necessitate secondary amputation for pain in the heads of the metatarsal bones, or in the metatarso-phalangeal joints, as in the case (one of Sir William MacCormack's) reported by Bland-Sutton,* who eventually performed amputation at the knee-joint. It has, however, the merit of including such a considerable mass of cancellous bone that recurrence of the disease is rendered very improbable. Of this operation Jacobson † says, "The result is obtained at far greater cost and risk than that by a Syme's amputation, and is, in my opinion, of very doubtful superiority."

Again, Watson's operation of almost complete tarsectomy, although it leaves the foot at a right angle with the leg, leaves a member which is not to be compared with a Syme's stump in point of utility, and is certainly more suited to cases of inveterate talipes than to those of tuberculous disease.

With regard to the hiding of the deformity, although a simple peg is the usual apparatus for hospital patients, it is possible at a small cost to fit an artificial foot to the stump, which outwardly bears an excellent likeness to the natural foot. The prospect of permanent cure is, if the case be taken sufficiently early, as here recommended, distinctly hopeful; and, indeed, the majority of cases show no further return of the disease in the stump, provided the heel-flap has not been allowed to become infected (as shown by the presence of sinuses or of the bluish purple patches which herald their appearance), the tendon sheaths are free from disease, and the ankle-joint is intact. And although one sees from time to time cases which have done well in spite of the latter having been affected, the result is probably only due to the greater facility for reaching the disease afforded by the complete opening up of the joint; one should not in such cases give anything but a very guarded prognosis.

The most important question from the patient's point of view is certainly the last. In the vast majority of cases the answer is an emphatic affirmative. There are few amputation stumps which provide so well for the patient's comfort and convenience as that of Syme's operation. Locomotion is free and rapid, and provided that no untoward complications arise, such as "painful stump," there are few occupations which cannot be followed by the patient; in fact, I am at the present time aware of a patient who, in spite of having parted with both of his feet as a result of a railway injury, can walk short distances well with the help of a stick, and thus carries on his daily work without much difficulty. Naturally such trades as involve the use of the tarsal and ankle-joints—such, for example, as turning, machining, or the riding of a bicycle—are much interfered with; but to a great extent this would have been the case after repeated erosion, partial tarsectomy, etc., the matting of the tissues having resulted in the production of a perfectly rigid and inelastic mass, useless for work, painful to walk upon, and unsightly to behold.

Mansfield Street, W.;
February 19th.

* 'Lancet,' 1893, vol. ii.
† Op. cit., p. 796.

The Spontaneous Absorption of Cataract.—At a meeting of German physicians at Cassel, Axenfeld, of Freiburg, gave a microscopical demonstration of a senile cataract, which was in the course of spontaneous recovery. The preparation was taken from an eye that had been affected for eight years. The patient was 82 years old. The cataract could be demonstrated by histological examination, like the case of Lindhal on the remains of a Morgagni's cataract, because the remains of the nucleus were found in the lower part of the empty sac of the capsule. Axenfeld concludes, with Nathanson, that spontaneous absorption is probably the rule in the Morgagnian cataract, if the cataract be left to itself, since in more than fifty cases reported this form of cataract is largely predominant. The final absorption depends on the softening of the cortex and of the periphery of the nucleus, and does not depend on a destruction of the epithelium of the capsule, which was supposed to be a necessary preliminary, for in this case and in the one reported by Lindhal, which was examined while undergoing absorption, the epithelium still remained to a great extent, just as in the Morgagnian cataract the epithelium is apt to remain.—*The Post-Graduate*, vol. xix, No. 1.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 592. WEDNESDAY, MARCH 2, 1904. Vol. XXIII. No. 20.

CONTENTS.

PAGE

*A Clinical Lecture on Giddiness. By FRED. J. SMITH, M.D., F.R.C.P. ... 305

*The Surgical Treatment of Gastric Ulcer. By A. W. MAYO ROBSON, F.R.C.S. 311

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

A CLINICAL LECTURE ON GIDDINESS.

Delivered at the London Hospital,

By FRED. J. SMITH, M.D., F.R.C.P.,

Physician to the London Hospital.

GENTLEMEN,—To-day I had a subject for our lecture suggested by the fact that in the last thirty-six hours no less than three cases of giddiness have come before me, and it occurred to me that on the subject of giddiness one could make a fairly useful lecture.

First of all it will not be a waste of time to consider what we mean by giddiness. I had a few minutes to spare this morning and I looked the word up in several works of reference that I have, but I could not find it. One at last suggested to me: "Giddiness, see vertigo." Well, that is right enough so far as it goes, but giddiness in ordinary language means more than vertigo. Under the heading of vertigo I found a certain amount of information which I did not want, because it entered rather too much into the details of localisation in the brain and the special influence that vertigo of fixed and definite characters had upon such localisation. That was too deep for my purpose in lecturing to junior students. In the term giddiness I include two classes of sensations: (1) this vertigo of a very definite character, due definitely to intra-cranial disease, and about which we shall speak presently when we come to the question of diagnosis; (2) a sort of indefinable sensation of unsteadiness, which is a very troublesome complaint common to all ages, one might say, and owning many different causes. Of the three cases which prompted this lecture there were two in men at or past middle life (one aged about forty and the other about fifty-seven), and the third case was that of a youth aged twenty-three. So you must bear in mind that giddiness

Digitized by Google

is really of two kinds—one a very definite vertigo with objects going round and round, or the patient appearing to himself to go round in certain directions, and the other in which he feels merely a sensation of unsteadiness or possibly faintness.

We will first go through the causes, because the real object of my lecture is to enable you to discover what is the matter with a given patient who comes to you with the main complaint of giddiness or unsteadiness.

First of all, it may be due to a definite cerebral cause, of which we may put down tumours and epilepsy as two typical examples. No doubt there are others. Secondly, it may be due to wrong impressions sent up to a brain which is healthy; for instance, those from the ear, the eye, or the skin may any of them be faulty. Thirdly, it may be due to vascular causes; of these cases there are two illustrative groups, viz. cases of simple fainting and those in which arterial degeneration plays a very great part. With regard to a fourth group, I may say there is a certain degree of doubt (it is not clear whether there is such a thing as a primary gastric giddiness), but there is said to be a group due to stomach derangements, *i. e.* to dyspepsia. The fifth group are what we may call toxic cases, the typical one being tobacco when taken to excess. Possibly some of you here remember when you smoked your first pipe and the giddiness it produced. There is still one more cause for giddiness which may be left as a separate group; that is to say, neurasthenia. So the groups of causes are: (1) definite cerebral lesion; (2) wrong impressions coming up to a brain which is in itself healthy; (3) a defect in the blood-supply to a brain otherwise healthy; (4) poisons brought by the blood to the brain and there affecting its cells; (5) stomach troubles possibly, and (6) neurasthenia.

Now we must see how to recognise to which group a given case belongs, in other words to make a diagnosis of the cause of the giddiness. We can do that in one of two ways; we can either take a group and point out certain signs which will be present if it is a member of that group, or we can make a systematic examination of the patient and draw conclusions as we go. I think it will be wiser perhaps to go through the causes and see what additional signs will establish the diagnosis.

Tumours of brain as a cause. If the giddiness is due to tumourit certainly is not likely to be the only complaint. A few inquiries will soon show that headache and probably vomiting are added; they are not only added symptoms but added distress, and will very likely be complained of voluntarily by the patient. With regard to the situation of a tumour which is most likely to have giddiness for a prominent feature, the middle lobe of the cerebellum is the most likely one. Now that same middle lobe of the cerebellum is the situation above all others for a tumour to cause headache, vomiting, and other symptoms of cerebral tumour. I might mention the reason for this: it is that beneath the tentorium cerebelli there is but little room, and therefore even a small tumour soon begins to cause pressure and the signs of tumour are intensified. Suppose, from the nature of the complaint and the way the patient spoke to you, a suspicion of cerebellar tumour were roused, the next step is to make the patient walk. If his giddiness were really due to cerebellar tumour, I think there is no doubt his gait would be markedly of the cerebellar or staggering variety, in which the patient swerves from side to side. If these points come out positively in the examination, you are very likely indeed to find the clinching symptom of cerebellar tumour present—namely, optic neuritis.

A more difficult cause to determine is the second one, namely, epilepsy, and for this reason: when giddiness is a symptom of epilepsy it is quite possibly the only symptom that is complained of. Of course if the man has had these attacks of giddiness for years and years interspersed with definite major fits, the diagnosis is made for you; but I am assuming that a man comes to you complaining for the first time in his life of giddiness. The giddiness may be the only sign of *petit mal*, or minor epilepsy. Therefore I say that the second cause, epilepsy, is an exceedingly difficult one to get at, because it is diagnosed mainly from negatives. On the positive side there may be a complaint that with the giddiness consciousness is lost; and if an attack of *gros mal* should have ever occurred to the patient there may be a history of a bitten tongue, of urine and fæces passed unconsciously, of sleepiness after the fit, and a family history to help you. But with these aids difficulties in diagnosis disappear. We will leave epilepsy for the moment, until we come to consider the matter from the other side.

Digitized by Google

Faulty impressions on a healthy brain—our second group. First, wrong impressions from the ear. Now, the ear, or rather the complete organ of hearing, consists, as you know, of three parts: the external ear, the middle ear, and the internal ear. And the internal ear is the part which is generally in trouble when giddiness arises from ear mischief, though wax in the meatus may cause it, and should be looked for. How are you to recognise the giddiness which comes on from middle-ear trouble? Well, there are one or two points which soon attract suspicion. First of all, the man's voice. I mention it first because it is the first thing you would hear, but I should have put it second after naming the cause of it, viz. deafness. Those of you who have a deaf friend or relative will not need me to explain what I mean when I say that the voice of a deaf person is a thing which can be recognised as belonging to a person who is deaf. The voice is sometimes raised, but not always; sometimes the man speaks more gently than usual, because his voice, although he is deaf, may sound louder to him than it ought to. You will thus probably at once say, this man is deaf, I wonder what it means, and you will naturally proceed to investigate his hearing power. In that investigation you are met with this difficulty, that the nerve to the cochlea, or that part of the internal ear by which we apparently become aware of external sounds, is supplied by a different branch of the eighth nerve to that which goes to supply the semicircular canals. Therefore, inasmuch as ear giddiness comes as much from the semicircular canals as from the cochlea, or more so, you may get the giddiness of internal ear trouble without any very definite deafness. However, it is comparatively rarely that the nerve to the semicircular canals is affected alone without any implication of the nerve going to the cochlea. Thus tests for deafness do become of importance. It would be going somewhat out of my immediate province to give you any deep or intricate tests for deafness, but there is certainly one which is rather striking and is most easily applied, and that is the ticking of a watch. You must see at what distance off he can hear the watch with either ear, and then place the watch on the patient's forehead so as to test his bone conduction. Now, if he is deaf in one ear and not in the other, there is the curious paradox that he will hear your watch thus placed on the forehead better in the so-called deaf ear than in the other. The explanation seems to me to lie in a matter of habit; he is accustomed to hear nothing with the deaf ear, so that when a sound does reach it the sound appears louder than usual. If you find that he is deaf, you will not have much difficulty in saying that it is a case of ear giddiness. As regards the actual causes at work in giddiness from disease of the internal ear, they seem to be one of about three or four groups. The first is Meniere's disease. Meniere described this condition a long time ago, and it was described first of all on symptoms only, without post-mortem evidence. When post-mortem evidence was forthcoming it was found in some of the cases that a hæmorrhage into the semicircular canals had taken place. Well, I say that a hæmorrhage into the semicircular canals should not be spoken of as Meniere's disease; it should be called "a hæmorrhage into the semicircular canals," detectable, if you like, by the giddiness. So there is first of all this hæmorrhage into the semicircular canals. An additional point here will be that the giddiness originally came on quite suddenly when straining, as at stool or on blowing the nose, etc. Secondly, there is syphilis affecting the softer structures of those canals. The only proof of that is this: that in some of these cases complaining of intense giddiness in which the tests show that the internal ear is at fault the patients get perfectly well under treatment by iodide of potassium and mercury. Then, thirdly, there is a sort of chronic sclerosis of the parts of the internal ear. Personally I think it is only to that chronic form, in which there is very little post-mortem change to be noticed with the naked eye, that we should apply the term Meniere's disease.

Now with regard to the wrong impressions from the eye. This is really a matter of false retinal impressions, and therefore indicates that there is a defect in one of the muscles concerned in perfect vision, for the muscles adjust the retina to a proper position. It may be one of the external muscles, bringing the case into the category of ophthalmoplegia externa; or it may be one of the internal muscles, the iris or the ciliary muscle (which are the main agents in accommodation), and then the case is one of ophthalmoplegia interna. How are you to suspect that it is the eye? A very simple and useful question early in the examination of a

Digitized by Google

case of giddiness is: "Is it worse or better with one or both eyes shut?" If better the case is almost certainly due to false retinal impressions as a primary cause. That is another point to be borne in mind as showing the necessity of making a systematic examination of a giddy patient. We have had to examine the eyes for optic neuritis, and now examination is necessary in order to detect whether there is double vision or not. If either double vision or nystagmus is present, the proximate cause is false retinal impressions. I have asked a student to test for nystagmus, and I find he holds his finger up before the patient and says, "Now watch my finger," and he wags it about here and there, showing that he has no idea of the test nor of what he expects to see. The proper way to test for nystagmus is to hold your finger or a pen up and say, "Watch this," and then move it slowly to one side, getting the patient to follow it with his eyes while his head is kept still; and when you have got your object so far out of the main visual axis that you know one external and the other internal rectus is put on the stretch, hold it there for ten or fifteen seconds. What nystagmus really means is a tiredness of the muscles; the jerkiness in their action is a consequence of this fatigue, and will only be shown if you tire them. I have carefully used the term proximate cause in these eye cases, for if the condition be not due to errors of refraction of congenital origin it is most likely to be ultimately due to gross disease of the brain, of which I have seen many examples. One I remember particularly, in which, by covering the faulty eye, the giddiness was at once cured; he died of cerebral tumour.

Now we will consider the faulty impressions coming to the brain from the skin. Here again you will see that we are getting a little bit out of what we may strictly call vertigo; we are dealing more with confusion of the senses than actual vertigo. Amongst these cases of false impressions from the skin there is no doubt that the typical example is our old friend tabes dorsalis, and you hardly need me to inform you how to test a man for that. But you might naturally ask me how would you get on the track of it. If a man were complaining of giddiness, I take it that if you have a suspicion of nerve troubles you should make him walk, and a tabetic walk will probably reveal itself. The next simple thing is to test his eyes again with a bright light, and if the Argyll-Robertson pupil is found then test his knee-jerks; if they are absent you need not go much further for a cause for his symptom. So much for wrong impressions from the skin.

Now with regard to vascular troubles. Here again, if you have been careful to ask your patient to describe to you as far as he can what he means by giddiness, and if he genuinely means that the room seems to be going round with him or that he is going round and the room is standing still, I do not think you need very much worry yourselves about it being a vascular lesion; the giddiness due to vascular degeneration is a confusion of consciousness, not a strict vertigo. In what sort of a patient do you find this? Practically they are always the type of man such as the one who first put the subject for this lecture into my head. He was about 55 years of age, and complained of nothing but unsteadiness and giddiness. He was a labouring man who looked his age, every minute of it; he was unhappy in his manner of speaking; he showed that he felt anxious and worried—his old appearance and manner put me on the track. I listened therefore to his chest, which is the place to get your first information from in any case of vascular degeneration. And what I heard there convinced me that he had a hypertrophied heart. His pulse was not exactly what you might call hard, but it was of good tension, rather above than below the average. The first sound of the heart was somewhat prolonged, and the second aortic was rather *plus*. You will notice that I do not say anything about where the apex-beat was. If with the stethoscope, after plenty of practice, you cannot distinguish a hypertrophied heart by the sounds and general impulse, the position of the apex-beat is not likely to help you; if you have to worry yourself as to where exactly the apex-beat is, you will come to grief. For in these people there is invariably associated with the vascular degeneration of their age a degree of emphysema which is almost always sufficient to obliterate anything like an ordinary apex-beat. That is a very different thing from obliterating a cardiac impulse, which is only to be appreciated by placing the whole of the hand on the chest, when a sensation is conveyed to the hand that there is a heavy organ working beneath it. Sometimes you get a heave of the whole of the chest wall, but not a trace of the apex-

Digitized by Google

beat. This heaving impulse or, perhaps better, impression, it was that put me on the track of this particular patient having vascular degeneration. You will perhaps ask what has that to do with giddiness? Why should not a man be old and have a hypertrophied heart and cirrhotic kidneys and emphysema without being giddy? And no doubt many such do not complain of giddiness. I admit that the explanation is purely hypothetical, but such as it is it has to satisfy you, because I cannot offer you anything more definite. If the giddiness depends to a large extent on this vascular degeneration, you will find that it is especially marked on stooping to do ordinary natural things, such as tying the bootlace, pulling up the stockings, or worrying yourself with your braces with the head down, or stooping down to pick up something off the floor—those are the sort of actions which produce giddiness. One's only conclusion, it fits in with the facts notwithstanding that it is hypothesis, is that when gravity thus acts upon the blood-vessels of the brain they are not sufficiently elastic to regulate that delicate tension in the capillaries which is necessary for the perfect working of the cortex. There is another cause for this disturbance in capillary pressure, and that is too low a blood-pressure. A person who has nearly bled to death, for instance, and has for a short time very low tension, will get giddiness on shifting his head about, and it is easy to see in that case that the tension is too low. In the other case it is too high for the moment, for the degenerate vessels cannot contract sufficiently rapidly to regulate the pressure, and so in either case the capillary pressure is deranged. If the tension is permanently low I think slight causes more easily disturb capillary pressure than when arterial tension is higher, and consequently attacks of faintness and giddiness are more likely to occur. This is the case in young subjects about puberty, in whom faintness and giddiness are rather common complaints. So much for the vascular group.

Now as regards the gastric group. I am bound to tell you that I found "gastric" mentioned as a group in a book of reference I had—I forget at the moment which it was,—but no cases were quoted, and doubts were thrown on its existence. I have only put it in here to remind me of the fact that dyspepsia has been alleged to cause giddiness. Certainly no definite case of it that I can remember has occurred to me. I could imagine that a man who suffered from dyspepsia could have such a congestion of his stomach under certain conditions that he would be giddy, owing to a deprivation of his brain of blood, the blood being required for the abnormal gastric functioning that was going on. Still, I have no experience of it. The only thing I would suggest would be this. You would ask the man, "When do you get giddy?" And he would reply, "I always feel giddy after a meal, so much so that I seem to get a little indigestion and have to sit down, or I should fall." I take it that is likely to be the history in a case (should you meet with one such) in which giddiness was due to indigestion from a gastric cause.

With regard to the next group, poisons, I mentioned just now the special one, which is within the experience of all of us, namely, tobacco, with which alcohol may also be joined. Tobacco is a very curious poison in this way, in that I doubt if any of us get thoroughly acclimatised to it. I admit that now and again towards evening I have found myself smoking too much; I have recognised the fact by an indefinable feeling of this obscuration of one's consciousness, which might perhaps by some be called giddiness. Even among those who smoke freely it seems always possible to get an excess of it. Certainly within my own circle of acquaintances I have known half a dozen at least who cannot smoke at all because they have discovered it gives them palpitation of the heart, and with that a degree of giddiness which was extremely unpleasant. Then, in what we may call the dangerous forms or causes of poisoning, there is almost invariably associated with the shock and collapse and the effect upon the heart and respiration a feeling of giddiness, especially if the patient tries to stand up or walk about. Speaking in general terms, I have no doubt there are in such cases two effects: (1) the specific effect of the poison on cerebral cells, and (2) the diminished capillary pressure owing to circulatory depression. I do not know any particular poison of which it may be said that giddiness is the special feature; I certainly do not call one to mind at this moment.

Now let us speak of the last group of all, neurasthenia. Neurasthenia in itself is an exceedingly difficult disease to attempt to define. It is an exceedingly difficult disease to satisfy *oneself* by the diagnosis. It is perhaps the easiest disease in

Digitized by Google

this world to diagnose to the extent of satisfying *the patient*, for they like the diagnosis. I will tell you what happened to me this morning. I saw a boy æt. 20, whose mother died a few years ago; she did not die in a lunatic asylum, though I do not say she ought not to have been in one. At any rate there was a family history of neurosis. This young man is without much will-power; that is to say, he is easily led astray. I do not know that he gets drunk, but he takes more whiskey than is good for him. He certainly goes in for fast living with women and loafing about; this I believe is entirely due to his inability to say "No," and is not a consequence of vicious inclinations in himself, but of weakness of character. Why he came to me was this: he gets up feeling pretty well, he eats his breakfast still in a healthy frame of mind, but directly he goes down to the station in order to come up to the City to work he begins to feel giddy, and when he gets into the train he says sometimes he almost loses consciousness, so giddy does he feel. And, by the way, I might have mentioned this "giddiness in a train" under false impressions derived from the eye. I believe every one of you can make yourselves giddy on a railway journey if you choose to do so, by steadily fixing the eyes on the bank and trying to abstract your mind from any purpose except the flitting by of the objects on the bank. With regard to this boy, it is possible that this may have something to do with it, but he said the sensation began almost as soon as he went to the train. He got it also in the office at times and, curiously enough, in church. What was I to say about that case? I had got the history, and therefore concluded that it probably did not owe its existence to any particular organic disease. The lad himself said that he was better when he went into the country, where he had no trains and no people to bother him. I examined him very carefully—his eyes, his ears, his knee-jerks—and made a few inquiries about indigestion. And there was just this to be said about it, that the lad did tell me definitely that he had a good deal of flatulent indigestion. Therefore it is possible that I may by accident have stumbled across a case that I said I had never seen, namely, gastric giddiness. Still, for all that, his manner, the tones of voice in which he talked to me, and the utter absence of any other cause to which one could reasonably attribute his giddiness convinced me that I had to deal with one of those unfortunate cases of neurasthenia; and I say unfortunate advisedly, because even when you have satisfied yourself that you are dealing with a case of neurasthenia, its treatment, which is the *raison d'être* of medicine, is so unsatisfactory. At any rate these features in the history and complaints convinced me that I had to deal with a man who was simply neurasthenic; in other words, if you like to call it so, and I think you may legitimately do so in this case, hysterical.

That gives you the leading features in each of these five groups. We will now work a case out from the opposite point of view, to see how in practice a diagnosis may be arrived at. Begin with the simple question, "What do you complain of?" Do not ask, "What is the matter?" for he will reply, "I want you to tell me that. I get so giddy." "When, *i.e.* under what circumstances, do you get giddy?" I think that is an exceedingly important question. Some are giddy only on walking about—cause, probably eye or cerebral lesion. Some, like this boy I have mentioned, get giddy only when they are in such places as a railway train; this comes very close to agaraphobia; that is to say, "frightened by being in a large space," which means neurasthenia at once. Some people become giddy on lying down in bed—probably vascular in causation; others when going to certain places; others on stooping—also probably vascular cause at work. Some, and this is important, get giddy when they blow their nose; remember that giddiness coming on when the nose is blown is almost conclusive evidence that the cause lies in the internal or middle ear, and you will at once have your suspicions directed to that. The patient should then be directed to walk away from and to you with eyes open and with them shut—from this many hints may be obtained as to the cause. Next you must proceed to make a systematic physical examination. I do not think there is any other way of finding out what the giddiness is due to. In this systematic examination you may begin where you like, and you will perhaps begin with those things which you think most likely to be present. For instance, you may begin with the eyes, for which of course you will want your ophthalmoscope. If the eyes are found to be normal you will proceed to examine the ears, and test their capacity with the watch or with the voice. The

Digitized by Google

human voice is not altogether and under all circumstances a good thing with which to test for deafness. The reason it is not good is because the overtones in a voice are so numerous and so complex that the patient may hear the overtones, and, therefore, know what you say, and yet all the time he may be deaf to the lower tones, and it may be to some of the higher ones also. But it is good inasmuch as it gives you a variety of sounds with which to test, and if the patient can hear any of them it shows that the hearing apparatus is not destroyed; moreover it is the sound which a patient probably wishes to be able to hear. If you use a tuning-fork there is only one note with its own special overtones going on. And again, the tick of your watch is a comparatively simple noise. Having examined the ears and eyes, you may then inquire about the stomach; ask about indigestion, whether the bowels are open regularly. I have known cases, which of course belong to the vascular group, where one hardly dare purge them because of the giddiness which came on when they were purged, thus pointing to a lowered tension in the blood-vessels, and, therefore, to a vascular cause of the giddiness. Then examine the chest, listen to the heart, feel the pulse. You have nothing much to do with the lungs, except in the cases in which when asked, "When are you giddy?" the patient says, "I am only giddy when I cough." It is much the same as being giddy when you blow your nose, only of course blowing the nose does not fill up the jugulars. In other words, coughing has a more general vascular effect than blowing the nose, the latter having a more special effect on the Eustachian tubes of the ear and, therefore, on the pressure in the middle ear. If you are satisfied that the heart and lungs and stomach are all satisfactory, you can then proceed to test the knee-jerks, though you will be likely to have done it in the earlier stages after seeing the patient walk. And now supposing everything is negative in the way of these organic troubles, your diagnosis then comes down to either epilepsy pure and simple, *petit mal*, or it comes down to a neurasthenic condition; and, as I have said, in the case which I had to deal with this morning that was my conclusion, owing to the fact that every possible organic cause was excluded, and then there was the family history to help me, but you now see why I said that epilepsy was so difficult to arrive at as the cause of giddiness—everything else has to be first excluded.

Harley Street, W.,
February 27th, 1904.

THE SURGICAL TREATMENT OF GASTRIC ULCER.*

By A. W. MAYO ROBSON, F.R.C.S.,
Hunterian Professor of Surgery and Pathology;
late Vice-President R.C.S. of England.

UNTIL quite recently gastric ulcer, except for one or two of its complications, has been considered to be a subject for medical treatment from first to last, and in ordinary cases very few surgeons, certainly none in Great Britain, have raised any question as to the desirability of continuing this practice. The profession is, however, becoming awakened to the fact that gastric ulcer is not the trifling ailment that it was once considered to be, and that it should from the first be taken seriously, for it is in the early stages that medical treatment can be employed to the best advantage, and in the later stages that general treatment is so often followed by relapse or by serious complications. I remember well when both cholelithiasis and appendicitis were always treated medically, and when it was generally believed that with very few exceptions neither disease had any mortality; but who would venture to uphold such doctrines in the light of more recent developments in diagnosis, pathology, and treatment? In the same way gastric ulcer, when I was a student, was looked on almost as a trifling ailment, and regularly treated in the out-patient department; but since we have become better acquainted with it and its sequelæ, it has been shown to be a really dangerous disease, not only serious from its direct effect on nutrition, but also from its very serious complications. One of the greatest medical authorities on the subject says that one half or three fourths of all cases will be cured by four or five weeks of treatment, but if not better in that time they will never be cured by medical treatment alone, a view in which I feel sure many of my audience will concur.

In considering the treatment of ulcer of the stomach it is useful to hold in view the course of an ulcer of the leg, which, directly the healing stage has arrived, becomes free from pain; but this neither indicates that healing is completed nor that care may cease, and should treatment be aban-

* Delivered before the London Polyclinic.

Digitized by Google

doned and the ulcer become chronic, though it may even then be painless, it is at any time liable to become inflamed or to extend; moreover the surrounding tissues become infiltrated with lymph which tends to organise, and this in a hollow viscus soon ends in stricture, as in the leg it tends to drag on the surrounding skin and produce constriction of the limb. Another simile may be found in ulcer of the rectum, in which no one would dream of trusting to general treatment after failure to obtain relief in the early stages.

Einhorn, in his well-known work on 'Diseases of the Stomach' (p. 223), says, "At first glance it would appear that the prognosis of gastric ulcer is quite good, especially nowadays, when the diagnosis of the affection is usually made at an early date." However, if we take into consideration the tabulated statistics given by Debove and Remond (p. 276) in reference to the outcome of all cases of ulcer, we become careful in our favourable predictions. This table gives in a hundred cases of ulcer—

Perfect cure	50.
Perforation and peritonitis . .	13.
Foudroyant hæmatemesis . .	5.
Pulmonary tuberculosis . . .	20.
Inanition	5.
Different complications . . .	7.

The most recent evidence we have concerning the results of medical treatment in cases of gastric ulcer is from the London Hospital, where Dr. Bulstrode collected and analysed the statistics of all the cases admitted for gastric ulcer from the beginning of 1897 to August, 1902. They were just 500 in number, 98 men and 402 women. Out of this number, 48 (nearly 10 per cent.) died from peritonitis, 13 (or 2·5 per cent.) from hæmatemesis, and 28 (5·5 per cent.) from other causes.

Equally interesting is the fact that out of the 500 cases, 211, or 42 per cent., were cases of relapsing ulcer; and of these, 116 had relapsed once; 41, twice; 15, thrice; and 39, four or more times. Mr. Mansell Moullin, commenting on these statistics, says, "Bad, however, as this sounds, it is not yet the worst. These figures do not include any of the cases admitted suffering from what may be called the late sequelæ and complications of gastric ulcer. They do not take into account the cases admitted for pyloric stenosis (which Gerhardt and Warren both estimate at 10 per cent.), hour-glass contraction, gastric dilatation, chronic dyspepsia, constant vomiting, perigastric adhesions, cardiac stenosis, and those numerous other disorders caused by the cicatrisation of ulcers healed long since. Nor do they take into account those cases in which carcinoma has developed at the site of an old ulcer, which Hauser estimates at quite 6 per cent."

When allowance has been made for all these serious complications of ulcers that have been treated medically, but which certainly cannot be described as cured, instead of 18 per cent. of deaths and the 42 per cent. of relapses, the numbers both of deaths and relapses must be much greater; and it cannot but be admitted that the latest statistics more than justify Leube's and Einhorn's estimate.

In discussing the surgical treatment of ulcerated stomach we have to consider not only the treatment of gastric ulcer itself, but also that of its complications and sequelæ, which are no less numerous than serious.

1. Acute perforation of the stomach wall and general peritonitis.
2. Hæmatemesis and melæna.
3. Cicatricial stenosis of the pylorus.
4. Hour-glass stomach.
5. Tumour of the stomach or pylorus of inflammatory origin.
6. Dilatation of the stomach due to obstruction.
7. Fistula between the stomach or pylorus and adjoining organs or with the surface of the body.
8. Spasm of the pylorus producing intermittent stenosis (Reichmann's disease).
9. Persistent vomiting.
10. Local peritonitis or perigastritis ending in adhesions.
11. Local peritonitis ending in suppuration and a localised abscess.
12. Subphrenic abscess.
13. Abscess of the liver, pancreas, or spleen.
14. Atonic motor deficiency.
15. Severe gastralgia.
16. Tetany.
17. Stenosis of the cardiac orifice.
18. Acute or chronic pancreatitis.
19. Profound anæmia resembling the pernicious form.
20. Pressure on or stricture of the bile-ducts with jaundice.

Digitized by Google

21. Catarrh of the gall-bladder from adhesions, producing attacks like those of cholelithiasis.

22. Great loss of flesh and strength, ending in phthisis.

23. Cancer secondary to ulcer. "Ulcus carcinomatosum."

As to whether our treatment should be medical or surgical, it will not be out of place again to refer to statistics; in fact, it seems to be the only rational method of comparison that can give us true data on which to act.

Now it will have been noticed that Einhorn accepts the rate of mortality as fifty *per cent.* for gastric ulcer treated medically, and Leube says that *only half or three quarters* of all cases of gastric ulcer will be cured by even the most thorough medical treatment. We thus see from these great medical authorities that general treatment alone is very far from satisfactory, and it is only natural, therefore, that the profession should carefully consider whether it can be content to let the matter rest in its present unsatisfactory state when surgery can hold out a prospect of relieving or curing a very large proportion of the cases that physicians confess are not amenable to general treatment.

It is for us to show what we as surgeons can do for these 50 per cent.—or, to take Leube's smallest estimate, 25 per cent. of cases,—which must be left either to suffer intermittently, or to become chronic invalids, or to die, unless surgery can do something for them.

For this purpose it would not be fair to take the surgical statistics of even three or four years ago, since the facts are very materially altered by the all-round improvement in operations on the stomach; and the contrast of 25 per cent. of deaths in cases treated medically and 5 per cent. in those treated surgically in the worst and most complicated cases, is so striking that I feel it incumbent to urge most strongly that, although cases of gastric ulcer should first be submitted to medical treatment, yet, if such treatment fails to cure in a reasonable time, or if relapses occur on the resumption of solid food, then medical should give place to surgical treatment; for it is unfair to the surgeon to hand over to him almost moribund cases, and it is unjust to the patients to persist in dosing them with medicine, or otherwise treating palliatively cases that can only be benefited or cured by surgical means.

It must not be lost sight of that the 25 per cent. mortality in cases treated medically includes all gastric ulcers, slight and severe, whereas the 5 per cent. mortality of cases treated surgically includes only the serious cases incurable by medical treatment, which we have reckoned as 25 per cent. of the whole; this, therefore, makes the contrast more marked.

We must not lose sight of the fact that surgery is only concerned with cases that medicine has failed to cure, and that surgeons must show something more than mere recovery from operation to claim success in any case.

As yet sufficient time has not elapsed for us to put in exact figures the number of relapses that have occurred after surgical treatment, though it is my intention to do this ultimately as far as my own experience is concerned. I can, however, give some general conclusions even at the present time, and this I propose to do under the different operations I am about to mention.

Operation.—Before the abdomen is opened it is quite impossible in many cases to say what operation or operations will be required, and the surgeon must be prepared to adapt himself to circumstances on discovering the position of the ulcer and the conditions associated with it, especially as to the presence or absence of adhesions and other complications.

Any one of the following operations, or a combination of them, may be called for in any individual cases:—Exploratory gastrotomy; gastro-enterostomy to secure physiological rest to the stomach and relieve the hyperchlorhydria, or, in other cases, to short-circuit a stenosis; excision of the ulcer; pylorectomy; pyloroplasty; gastroplasty; gastro-gastrostomy; gastrolysis; pylorodiosis.

Gastrotomy, for the purpose of exploring the interior of the stomach, is an operation occasionally called for in the surgical treatment of ulcers.

(*a*) In order to verify the diagnosis of ulcer when there is so much thickening of the stomach walls as to suggest the presence of cancer. In a case of this kind that came under my care recently, and which I will describe more fully later, the symptoms all pointed to cancer, and on exposing the stomach a tumour was discovered in the lesser curvature which both my colleagues and I thought might be malignant; yet on opening the

Digitized by Google

stomach and passing the finger upwards it immediately entered a deep crateriform chronic ulcer forming the centre of the tumour, which was clearly inflammatory and curable by excision, as proved by the sequel.

(*b*) When, although the symptoms have pointed to ulcer as the cause of the gastric trouble, the stomach, on exposure, betrays no evidence of puckering or other characteristic signs; and when, in order to verify the diagnosis and ascertain what is best to be done, it is felt desirable to examine the interior of the organ, as in the case of a man æt. 38, who had had symptoms of chronic ulcer extending over several years. On exposure of the stomach by abdominal section no evidence was found on the surface to indicate the accuracy of the diagnosis. Exploratory gastrotomy was performed, leading to the discovery of a large chronic ulcer, one and a half by three inches, on the posterior wall of the stomach, adherent to the pancreas, and clearly not capable of safe removal. A posterior gastro-enterostomy was done, and recovery followed with complete restoration to health. In other cases where there is nothing to see on exposure of the stomach, on exploring the interior by means of gastrotomy, ulcers have been discovered and rational treatment has then followed.

(*c*) In certain cases of gastrorrhagia it is desirable to perform exploratory gastrotomy in order to find and ligature the bleeding vessels, or to otherwise arrest the hæmorrhage. In a paper I read before the Medical Society (see 'Lancet,' December 13th, 1902) on the "Surgical Treatment of Hæmatemesis," I related several cases of gastrotomy for the discovery of the source of the bleeding. In those cases, as in others, gastrotomy has enabled the bleeding points to be discovered, and has made the subsequent treatment a matter of certainty rather than a haphazard procedure, since it has shown what cases were suitable for excision of the bleeding ulcer, what were likely to be cured by simple ligature *en masse*, and what might be left to the rest cure by the performance of gastro-enterostomy.

(*d*) It necessarily forms part of any operation for the excision of ulcer of the stomach.

I do not propose to enter into any detailed description of the operation, which requires to be varied according to the object in view.

Excision of the ulcer is an operation strongly advocated by some surgeons, and in certain cases it may be no more difficult or dangerous than a simple gastro-enterostomy—for instance, where the ulcer can be readily exposed, and where it is not adherent to any of the surrounding viscera, such as the pancreas. Under such circumstances I have excised a gastric ulcer on nine occasions, recovery having followed in each case.

But where there is a difficulty in exposing the ulcer, and where it is adherent to other organs, I would decidedly advise a gastro-enterostomy alone as being simpler and safer, and, as a rule, quite as efficient. When it is borne in mind that ulcers are frequently multiple, and that, although one may be excised, yet the undiscovered ulcer may be actually the one producing trouble; and when it is realised how difficult it is to reach and excise an ulcer close to the cardiac orifice, even if it be discovered, or how almost impossible it would be to remove some of the large chronic ulcers of the posterior stomach wall, so often adherent to the pancreas; I think those who have had experience will agree with me as to the desirability of performing the less severe operation, even if it appear to be less scientific than the complete removal of the disease.

As illustrating the difficulty that may attend excision of ulcer, I would refer to the description of a case of combined scraping and excision published in the 'Lancet,' June 8th, 1901, in which, although the patient recovered, some months subsequently a gastro-enterostomy had to be done, which resulted in a complete cure.

Even in cases where I have excised the ulcer I have considered it advisable also to perform gastro-enterostomy, in order to give the stomach rest and to secure healing of the stomach wound, as well as any possible remaining ulcers.

After excision of an ulcer the bleeding from large vessels must be controlled by ligature, but the oozing from smaller vessels will be stopped readily by the continuous suture employed to bring together the edges of the wound. If the excision involve the serous coat, a Lembert's continuous stitch with a silk or celluloid suture will be necessary. Should the excision have been near the pylorus, the line of suture must be placed transversely to the axis of the canal so as to avoid stricture, as in a case of a woman æt. 43, on whom, on July 9th, 1902, I performed complete

Digitized by Google

excision of a pyloric ulcer, and was able to bring the edges so cleanly together in a direction transverse to the axis of the stomach that I did not think it needful to do a subsequent gastro-enterostomy; and in the case of a man on whom I operated on November 15th, 1900, who had two ulcers at the pylorus, one on the anterior and one on the posterior wall, both of which I was able to excise in a similar manner. In each of these cases no relapse has occurred, and as in the one nearly three years have elapsed, and in the other one year since the operation, the method seems to have been justified. The following example, previously referred to, is given in detail, in order to illustrate a method of performing excision which I have found simple and efficient.

Mr. S—, æt. 55, who had suffered from indigestion for several years, began to have severe pain about an hour after food, and to lose flesh and look ill, during the six months before I saw him. A doubtful tumour could be recognised, and during the attacks of pain the stomach could be felt to harden under the hand. Free HCl was present after a test meal. The serious nature of his ailment was brought home to the patient by his inability to continue his business, and, on the advice of his medical man, I saw him, and supported the opinion as to exploration. On operating I found the swelling to be a greatly thickened pylorus, forming a distinct tumour, and that about an inch and a half from the pylorus, along the lesser curvature, was another tumour, which, from the enlargement of the glands, we suspected might be growth; but on opening the stomach it was found that the pyloric swelling was an enormously hypertrophied sphincter, and that the stomach tumour was thickening around a chronic ulcer. The latter was freely excised by everting the portion of stomach involved and grasping it by means of the rubber-covered blades of my gastro-enterostomy clamps, which effectually controlled all bleeding. An elliptical incision was then made, so as to include the ulcer in its whole thickness and extent. After excision the sides of the wound in the stomach where the ulcer had been were brought together by two continuous sutures, the first embracing the muscular coat and the second the mucous edges. When the clamps were removed there was no bleeding from the sutured wound. The stomach opening was closed, and a posterior gastro-enterostomy was then performed. As the gall-bladder was full of thickened mucus and gall-stones, it was at the same time explored and drained. A smooth recovery has been followed by restoration to health.

As showing the greater severity of the operation of excision, Peterson and Machol ('Brit. Med. Journ.,' Suppl. October 11th, 1902) did seven excisions of ulcer with three deaths, and four resections of the pylorus for simple ulcer with two deaths—a very high mortality when compared with the same authors' results in gastro-enterostomy, eighty cases with five deaths.

Rydygier prefers excision of the ulcer to gastro-enterostomy, because he believes that carcinoma not infrequently develops in the scar of an old ulcer; but while accepting the fact that chronic ulcer predisposes to the development of cancer, I think it has yet to be proved that a soundly healed scar does.

Pylorectomy in chronic ulcer of the pylorus is a method for the more complete excision of the ulcer, and has usually been performed by mistake, the thickened pylorus having been thought to be malignant; but in some cases it has been undertaken because complete removal of the pylorus has appeared to be more simple than excision of a chronic ulcer in this situation. The operation may be completed either by an independent gastro-enterostomy, or by joining up the duodenum to the open end of the stomach.

As pylorectomy under these circumstances is much more limited in extent than in malignant disease, it is quite easy to make an end-to-end apposition; and in order to expedite the operation, and to be certain that the lumen is left sufficiently patent, the use of a bone bobbin presents to my mind considerable advantages.

Dr. Rodman ('Philadelphia Medical Journal,' June 9th, 1900) collected from literature and personal correspondence, detailed reports of forty pylorectomies, partial gastrectomies, and excisions for ulcer, with six deaths, or a 15 per cent. mortality. This included cases since 1881, but later operations under improved technique contrast favourably with the earlier ones.

Nevertheless the mortality after excision will probably always be higher than the more simple operation of gastro-enterostomy, and the more severe operation of pylorectomy should, therefore,

Digitized by Google

be reserved for cases that are not suitable for the less severe procedure, or in which the suspicion of cancerous degeneration is entertained, and cannot be disproved on naked-eye inspection. I have only once performed pylorectomy for simple ulcer, and in that case the tumour formed by thickening around the ulcer gave rise to a suspicion of malignancy.

It must be borne in mind that a tumour of the pylorus due to chronic ulcer is usually firmly adherent to the adjoining viscera, and that the operation will be correspondingly difficult, more so than in the early stages of a cancerous tumour, which is usually free. In several cases I have opened the pylorus and found an ulcer which I could excise, and I have then stitched up the longitudinal wound in the pylorus transversely, as in pyloroplasty. This is a simpler operation than pylorectomy, but it should, if possible, be followed by gastro-enterostomy, on account of the tendency of any wound of the pylorus to contract and so lead to pyloric stenosis, which will probably demand a gastro-enterostomy later.

Gastro-enterostomy, in the absence of special complications, is the operation to be relied on in the treatment of ulcer of the stomach; it acts by securing physiological rest by means of drainage, thus allowing the ulcer to heal without being subjected to the irritation of acid secretion, accumulation of food, or frequent stomach movement. It also, while remedying the hyperchlorhydria, relieves pyloric spasm, and, while preventing stagnation of fermenting fluids, materially diminishes gastric dilatation. The posterior operation is the one I personally prefer, the junction of the posterior wall of the stomach with the first part of the jejunum being effected by two continuous sutures, with or without a decalcified bone bobbin. The use of a bone bobbin not only secures an ample and immediately patent opening between the two viscera for the passage of the stomach contents, but protects the line of union from irritation.

The whole operation can be easily completed in half an hour, and it may even be done in half the time. An extensive experience with the posterior operation has been favourable, not only in the rate of recovery of the patients, but in the smoothness of the recovery, many of the patients recovering without even once vomiting; and only very rarely have I seen regurgitant vomiting of bile, which in the anterior operation is much more frequently seen, and at times becomes serious, or even leads to a fatal issue. Up to the end of last year I had performed the posterior operation on 103 patients with four deaths, or an average mortality of 3·8 per cent.; and this includes every patient submitted to this operation, without any selection, the greater number of the patients being extremely ill at the time of operation.

The most brilliant results occur where there is pyloric stenosis due to chronic ulcer, and in those cases where the ulcer has led to the formation of a well-marked tumour, whether at the pylorus or in the body of the stomach, as also in hour-glass contraction, and in all cases where the ulceration has produced well-marked and obvious damage to the stomach walls.

The least satisfactory cases are those in which the nervous symptoms have predominated, and where the pylorus is patent. In three of the latter class of cases I have been much disappointed with the ultimate issue, the patients having made a good recovery from the operation, but having complained subsequently of pain after food, or having had occasional vomiting, anorexia, or other untoward symptoms such as were present before the operation. I have reported so many examples illustrating the treatment of gastric ulcer by gastro-enterostomy, that I must limit myself in this lecture, on account of time, to references to cases in 'Diseases of the Stomach and their Surgical Treatment,' published by Messrs. Baillière, Tindall, and Cox, 1902; and to lectures published in the 'British Medical Journal' for May 25th, 1901, in the 'Lancet' for February 28th, 1903, and the 'British Medical Journal' for April 25th, 1903. I will, however, refer briefly to the after-histories of some of the cases where gastro-enterostomy has been done.

Mrs. W—, gastro-enterostomy performed for pyloric stenosis, due to ulcer, in November, 1898. In October, 1899, the patient was perfectly well, and had gained 1 st. 10 lbs. in weight. When heard of last year she was in good health.

Mrs. M. A—, æt. 28, gastro-enterostomy performed in August, 1899, for pyloric ulcer with great thickening of the pylorus. A year later she had gained 1 st. 4 lbs., and when heard of in 1901 she was in very good health.

Mr. D. B—, æt. 31, operated on in May, 1898,

Digitized by Google

for extensive ulceration of the stomach associated with inflammatory tumour. Two months later he had gained 1 st. 3 lbs. in weight, and when I saw him in May of this year, five years later, he was in perfect health.

Miss H—, æt. 32, gastro-enterostomy performed November, 1900. A report in 1901 stated that she had gained 1 st. 8 lbs., and was completely restored to health.

Mr. H—, æt. 52, operated on in November, 1900. Report received in March of the following year to say that he had recovered his former weight and was well.

J. S—, æt. 45, operated on in June, 1900, for ulcer of the pylorus. When heard of a year later was quite well.

Miss N—, operated on in April, 1901, for multiple ulcers. When seen in 1903 she was in perfect health, leading an active and useful life.

Miss A—, æt. 34, operated on in July, 1902. Letter received from her medical man a month ago to say that she was in perfect health, and had gained 3 st. in weight.

This list could be very much amplified, but these after-histories will suffice to show that it is not merely relief, but cure that may be expected to follow in the greater number of cases.

Pyloroplasty as a curative measure in this class of cases has certain very definite limitations, though it can be performed rapidly and with very little exposure of viscera. If the pylorus be stenosed, free from extensive adhesions, easily drawn forward, and not actively ulcerating, it is a simple and short operation, and in quite a number of cases of both gastric and pyloric ulcer I have found it to answer well. It must not be relied on, however, where active ulceration of the pylorus itself is found, unless at the same time the ulcer be completely excised; otherwise cicatricial contraction will follow. It acts in the same way as gastro-enterostomy, by affording a free exit to the stomach contents and thus securing physiological rest to the stomach. Pyloroplasty is too dangerous in cases where there is extensive induration of the tissues, much peripyloritis, and adhesions to the liver, gall-bladder, colon, and pancreas; and in cases of duodenal stenosis it is, of course, useless. It may be indicated in cases of spasmodic stenosis and in slight annular stenosis from ulceration accompanied by muscular hypertrophy, but even here I think that gastro-enterostomy is the better operation.

Although in my earlier operations on the stomach I thought favourably of pyloroplasty, along with other surgeons I have had reason to modify my opinion of the operation, not only on account of contraction of the new orifice, but from adhesions forming over the site of the sutured pylorus, which have a tendency to anchor it and to interfere with the stomach functions, although the canal may remain patent. I therefore now perform gastro-enterostomy by preference.

In a lecture given before the Oxford Medical Society, November 14th, 1902, and published in the 'Lancet,' February 28th, 1903, I related a case in which pyloroplasty was performed in 1895, but in which, on account of a recurrence of symptoms, I had subsequently to perform a gastro-enterostomy; and the same sequence has occurred in two or three other cases.

Nevertheless I can point to a number of patients, now in good health, who show that such untoward results do not constantly follow this operation, and that the after-results may be all that could be wished for. In 'Diseases of the Stomach and their Surgical Treatment,' published in 1901, can be found a report of a number of cases proving this statement. The following are examples:

Mr. B—, on whom I performed pyloroplasty in February, 1900, has had no further trouble, and remains quite well three years later.

Miss T—, æt. 45, had pyloroplasty performed in August, 1901. When seen quite recently she had more than gained her lost weight, and was perfectly well.

M. B—, operated on in September, 1901, was quite well this year.

The more complete operation suggested by Dr. Finney may perhaps have better ultimate results than the old method, but as yet sufficient time has not elapsed to prove it. Though the mortality in 318 cases collected from all sources at the time I gave the Hunterian Lectures was 15·4 per cent., I feel sure that properly selected cases from all sources would at the present time yield a very small death-rate; for instance, out of twenty-one cases of pyloroplasty that I have myself performed since 1896 there has been no fatality.

Gastroplasty is an operation that I have successfully employed in a number of cases of chronic

Digitized by Google

ulcer leading to hour-glass stomach. It consists in making a longitudinal incision through the strictured part of the stomach and bringing the edges of the wound together transversely, thus obliterating the stricture. A convenient method of performing the operation is by the use of a large decalcified bobbin. If the strictured part of the stomach be actively ulcerating, the ulcer must be excised at the same time; otherwise subsequent contraction may occur, or possibly the ulcer, already chronic, may persist and lead to a continuance of the symptoms; in such a case, if excision be impracticable, gastro-enterostomy must be performed, or if the pylorus be free from disease the operation of gastro-gastrostomy may be done in order to short-circuit the constriction.

I have operated on nineteen cases of hour-glass stomach due to chronic ulcer, with seventeen recoveries. These cases have yielded excellent results, and the patients rapidly gained flesh and strength. The case of gastroplasty that succumbed was suffering at the same time from pyloric stenosis, so that a double operation had to be done. Had I another case of this kind I should now at once perform posterior gastro-enterostomy, and so save all the troubles that were associated with the detachment of extensive adhesions before gastroplasty and pyloroplasty could be effected. In the light of subsequent events it is easy to see that a mistake was made in trying to do an extensive plastic operation in the presence of such seriously damaged tissues.

The after-histories of some of these patients are very remarkable. One lady, who had been an invalid for many years and who was worn almost to a skeleton, made a rapid recovery, and when I saw her recently she was between 3 st. and 4 st. heavier than at the time of operation.

Another, operated on in June, 1899, in which the stomach had three cavities, gained over 2 st. in weight, and remained well three years later.

An almost similar description applies to the other cases, except one, where pyloric adhesions of great severity necessitated gastro-enterostomy about a year later, which operation was followed by complete cure of all stomach symptoms.

Gastrolysis.—Adhesions of the stomach to adjoining organs are so common in chronic stomach ulceration that gastrolysis, or the detaching or otherwise treating bands and short adhesions to adjoining viscera or to the abdominal wall, is performed in by far the greater number of cases. Such adhesions are frequently only the remnants of ulcers that have healed; at other times they have been left by perforation of the stomach wall by an ulcer, from the direful consequences of which they have saved the patient. In many cases they give rise to symptoms resembling ulcer, though the adhesions may be due to causes, such as gall-stones, outside the stomach itself; in such cases the operation of gastrolysis may be entirely curative. I have performed gastrolysis in a large number of cases, all of which, when this was the only procedure required, recovered; in others the gastrolysis only formed a minor part of the operation, and gastro-enterostomy, pyloroplasty, cholecystotomy, etc., had to be done at the same time, the stomach dilatation being a secondary affection.

In a great number of cases on which I have operated for gastric ulcer I have found separate adhesions at six or eight places on the surface of the stomach, both on the anterior and posterior wall, showing where ulcers had been, and how, wherever they had approached the peritoneal surface, a localised adhesive peritonitis had occurred. Such adhesions, being often slight, are easily detached. In others, as after extensive ulceration of the pylorus or after such an operation as pyloroplasty, the adhesions to the liver or to the parietes are so extensive that, although it is possible to detach them, it is quite manifest that they must speedily re-form. In some such cases I have successfully grafted omentum over the new surface, but I feel sure that under those circumstances it is better to perform a gastro-enterostomy; for although the pylorus may be patent, yet where it is held up to the liver or parietes and fixed there rigidly, the effects on the motor function of the stomach are very marked, and I have seen patients in this condition suffer excruciating pain until relieved by the short-circuiting operation.

The most marked example of the beneficial effects of simple gastrolysis occurred in a patient on whom I operated in February, 1900. Miss B—, æt. 42, had been an invalid for several years. When I exposed the stomach, expecting to find a stricture of the pylorus, I only discovered adhesions binding the pylorus to the liver. After the operation she completely lost her pain and indigestion, and from being a chronic invalid, constantly requiring sedatives to relieve her distress, she

Digitized by Google

rapidly gained flesh and strength, and when I saw her last year she was stout and well.

Pylorodiosis, by which name is understood the operation of stretching the pyloric sphincter—either by means of the fingers invaginating the stomach wall, when it is known as "Hahn's operation," or by digital or instrumental stretching after having made an opening into the stomach, when it is known as "Loreta's operation,"—is a method of little practical value in the treatment of ulcer; and in some of the cases where I performed the operation, though the immediate results were good, relapses subsequently occurred. If performed by invagination without opening the stomach cavity, it is an operation unattended by risk. It may possibly be of service in simple spasm of the pylorus, but I have not much faith in its effects being lasting, for to stretch the pylorus to an extent sufficient to paralyse the sphincter must mean that the mucous membrane or even the other layers of the pylorus become lacerated, and thus will in due time lead to chronic ulcers. In two cases I have felt this to occur, and have laid open the pylorus immediately and found the laceration still bleeding; and in two other cases I have subsequently had to perform gastro-enterostomy on account of recurrence of symptoms.

As we are all agreed with regard to the desirability of adopting surgical treatment at the earliest possible moment in cases of perforated gastric ulcer, as well as in cases of suppuration in connection with ulcer of the stomach, I do not propose to consider these cases in the limited time to-day.

Indications and contra-indications for operation.—Having shown that there are many cases in which, after the failure of medical treatment, surgery is capable not only of obtaining operative success, but genuine cure, and having mentioned the operative means by which the surgeon may hope to bring about such cures, we may now profitably consider under what circumstances the surgeon may hope to do his best work.

We need not speak of acute perforation with general peritonitis, nor of chronic perforation leading to abscess, subphrenic or otherwise, for in these cases there is no room for two opinions—they are surgical, and surgical alone.

Although medical treatment, lavage, and careful dieting may for a time do good in pyloric stenosis consequent on ulcer, I have seen and operated on many cases where this treatment had been carried on far too long, the patients being handed over to the surgeon in a state of profound exhaustion. Could we see them at an earlier period, very little risk would attend operation, and there is no reason why these cases should have any mortality.

I can point to a considerable number of such patients, now living useful and active lives, who had for years been existing on the verge of starvation. In these cases, therefore, I urge that surgical treatment should be sought much earlier. The same remarks apply to hour-glass contraction of the stomach, which, when advanced, produces symptoms almost like those of pyloric stenosis. It is quite clear here that medicine can do no permanent good, but that surgery holds out great prospects of success.

In cases of perigastritis ending in pyloric adhesions, or in adhesions of the stomach to the abdominal wall, which so often complicate ulcer of the stomach and lead to severe and persistent gastralgia, made worse on movement, to inability to take food, or to vomiting after it has been taken, and to other untoward symptoms, it is quite clear that general treatment can do no permanent good, for the trouble is mechanical. These cases should be treated surgically, for the operation of gastrolysis, as I have shown, can be performed with very small risk; and even if, as is necessary in some of the cases with extensive adhesions, gastro-enterostomy has to be performed, the risk is well under 5 per cent. Whatever may be our views with regard to acute hæmorrhage threatening life, I think that very few among us, whether physicians or surgeons, can have any doubt about so-called chronic hæmorrhages, which are repeated over and over again, and which ultimately end in profound anæmia. These cases ought to be operated on before the bloodlessness is carried to such a state that operative treatment becomes dangerous.

I would not here express my own opinion with regard to acute hæmorrhage too strongly, as I have already given my views at considerable length before the Medical Society. I think, however, one should distinguish between the hæmorrhage that occurs in young anæmic girls, and which was described originally by Dr. Donald Hood (Medical Society of London, February 15th, 1892), and afterwards by Dr. Hale White, which cases usually end in recovery, and the violent hæmorrhage that

Digitized by Google

accompanies gastric and duodenal ulcer, which is usually repeated, and in a considerable proportion of cases ends fatally. My own view is that these latter cases, when recurrent, require operation for the arrest of the bleeding, and it may be undertaken, as I have shown, with great probability of success.

In another class of cases, where chronic ulcer is associated with tumour which simulates cancer, and where, even when the abdomen is opened, it is hard to tell whether the disease be simple or malignant, gastro-enterostomy may be completely curative, as I have amply proved by a series of cases, some of which were published in the 'British Medical Journal,' April 25th, 1903, when considering the treatment of cancer of the stomach. Several of these cases, now absolutely well and in perfect health, had been condemned as hopeless.

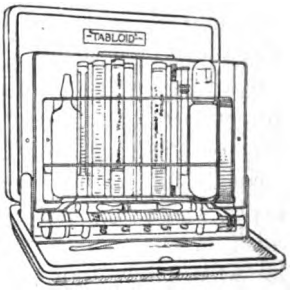
But when we come to consider operative intervention in cases of rebellious dyspepsia, in hyperchlorhydria, in nervous dyspepsia, in atonic dilatation of the stomach, and in other functional diseases, the surgeon is treading on dangerous ground, for he has to consider not only the risk to life in cases that would not end fatally, but also the possibility or even the probability of relapse. The want of clinical success, to say nothing of the disappointment to the patient, which such operations if indiscriminately performed would probably show, will be likely to bring discredit on a branch of surgery which is making sure and steady progress, and which is likely to have still greater triumphs in store.

As the result of experience in some of these cases, both in my own practice and in that of other surgeons, I would urge most strongly that every case of suspected ulcer of the stomach should first be submitted to thorough medical treatment, and only after failure of therapeutic measures should the question of surgical interference be entertained. But even with the greatest care there may still be cases of failure in diagnosis, and an exploratory operation advised. In such cases, if the physician feels that medical treatment has been carried out for a sufficient length of time without success, and that he is powerless to do more, recourse may be had to an exploratory operation, but even if after exposure of the stomach no evidence of organic disease is obtained, it may be wise to abstain from further intervention; for it must be borne in mind that nervous troubles frequently manifest themselves in the shape of gastric ailments, and that in some cases gastric ulcers, though completely cured by medical treatment, may have initiated a state of neurasthenia leading to a persistence of the original symptoms which cannot reasonably be expected to yield to surgical treatment except through its effect on the imagination.

It will thus be seen that while I advocate most strongly the surgical treatment of ulcer of the stomach and many of its complications in properly selected cases, I feel that the surgeon and physician should in all cases work together; and while on the one hand a medical colleague should not persist in treating cases of organic disease of the stomach so long that when handed over to the surgeon they are almost or quite unfit for operation, on the other hand it behoves the surgeon to be careful not to take in hand any case in which medical treatment ought to have a fair trial before he interferes.

Park Crescent, W.,
February 26th, 1904.

THE Hypodermic "Tabloid" Brand Pocket Case (No. 21) is now issued in gun-metal, so that medical men who prefer an aseptic hypodermic outfit may avail themselves of this useful and extremely compact case. The outside measurements are $3\frac{1}{2} \times 3\frac{1}{8} \times \frac{3}{4}$ inches, and the case may there-



fore be carried in the waistcoat pocket. The fittings comprise a special detachable nickel-plated aseptic frame and revolving rack for nine tubes of "Tabloid" hypodermic products, capsule of ether, glass-stoppered and capped phial, the Burroughs Wellcome and Co. All-glass Aseptic Hypodermic Syringe, with one exploring and two regular needles.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 593. WEDNESDAY, MARCH 9, 1904. Vol. XXIII. No. 21.

CONTENTS.

PAGE

*Two Lectures on General Paralysis of the Insane. By GEORGE SAVAGE, M.D., F.R.C.P. LECTURE I 321

*Proliferating Papillomatous Ovarian Cysts. By E. STANMORE BISHOP, F.R.C.S. 329

The Results of Lorenz's Treatment of Congenital Displacement of the Hips 335

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, *Holborn, E.C. Telephone* 1717, *Holborn.*

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for Binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½, *Bartholomew Close, London E.C.*

TWO LECTURES
ON
GENERAL PARALYSIS OF THE INSANE.*

By GEORGE SAVAGE, M.D., F.R.C.P.,
Consulting Physician to Guy's Hospital; formerly Physician to Bethlem Royal Hospital.

LECTURE I.

GENTLEMEN,—We are going to consider to-day varieties or, if you like, divisions of general paralysis of the insane. I suppose the first thing is rather to consider whether general paralysis of the insane deserves to be considered as a specific disease; that is to say, a clear and separate entity. One feels perfectly certain that the time will come when there will be a more satisfactory pathological basis for this group of diseases. One thinks of one's old teacher, Sir S. Wilks, who used to say, "I come round Bethlem and I see the most active patients—the men who rush here, there, and everywhere—and you name them prophetically paralytics." There is of course a great deal of truth and a good deal of Wilks in the statement. The fact that the termination is paralysis has to be admitted, but that the disease should and will later be named as progressive paralytic dementia, a general paralysis followed by dementia, or by any very similar terms, I do not think. It is undoubtedly a degeneration and something more, and in my last lecture I shall describe other forms of syphilitic degeneration which are met with in the nervous system. Just at this moment I was talking to Mr. Jonathan Hutchinson, and he said, "I suppose you look upon general paralysis still as distinctly associated with syphilis?" "Yes, certainly. But one has some other considerations to take into account." There are whole tribes, I

* Delivered at the Medical Graduates' College and Polyclinic.

Digitized by Google

believe, of Arabs and other semi-civilised people who have come into contact with civilisation and also with syphilis, and where syphilis is widespread, and yet among such it is said that no general paralysis occurs. One had, I remember thirty odd years ago, a rather strong view that there might be something racial in it, and, together with another physician who was connected with one of the large asylums, I investigated the occurrence of general paralysis among the Celts. The Irish were free from it, we heard, and also the Welsh in the mountains, and likewise in Cornwall the people were free from general paralysis of the insane. Therefore one was inclined then to think that the disease might have something to do with the race. But on investigation one found that it was no more true that the race had anything to do with it than it was true that black blood prevented men from having it. I remember when in America having heard it was almost unknown amongst the blacks, and it was said that it was unknown among the negroes as long as they were slaves, but that afterwards, when they became mixed with the world at large, they suffered as much as, if not more than others. In the same way it used to be said it was a disease purely of men. Then afterwards it was said that it was a disease occurring occasionally in women, but never in the cultured classes. I remember Maudsley years ago saying he had never seen a case of general paralysis occur in a well-bred woman. But I think that now women have claimed and asserted their rights to wider work and liberty they have had to incur some of the penalties. At all events I have seen more general paralysis among women in the last ten years than I did in the first ten years of my professional work. General paralysis, then, has to be considered at present as a convenient group of cases that have many similarities, the chief characteristic being a progressive nature that ends in paralysis and death within a comparatively limited time. One other question is, Is there such a thing as general paralysis, or progressive paralysis without mental symptoms at all? I used to doubt whether there was. Then one had to admit that there were some cases in which the mental symptoms were so slight and were so passive, if I may use the term, that they were passed over. I shall have to refer to one form in which one gets simply progressive dementia without any excitement, without any stage of mania, melancholia, or the like. I have known patients of this kind, as, for example, a man with the usual history: a middle-aged married man who many years before had had syphilis, with comparatively slight constitutional symptoms, was seriously concerned because his speech was becoming rather thick. His profession rendered it necessary that he should be a speaker, and the consequence was that when it became blurred and people could not hear what he said he had to employ clerks and others to do a good deal of his work. He came to me under those conditions, there being absolutely nothing in the way of acute mental excitement or acute melancholiac manifestations; in fact, there were no mental symptoms at all. For several years that man came once or twice in the year to see me, his speech becoming more and more affected, his handwriting getting more and more shaky, his defect of expression becoming more and more marked, his pupils becoming more definitely unequal, and so he went on. But in the end he had some epileptiform seizures, then progressive mental weakness developed itself, and the man died paralytic. But for one or two years that man, having most of the symptoms of general paralysis yet had no evident defect of mind, so that he was able to fully pursue his profession up to almost the time of his having a fit. We have, then, got to deal with a disease which I should say sooner or later, if the patient live long enough, develops some mental defect. But there are cases, and I have seen several, in which there have been symptoms similar to those I have just mentioned, in which the man has had one fit and that a fatal one, so that the man has had the changes going on in one part of the brain that has ended in a very severe form of epileptiform or congestive attack, and the man has died before mental symptoms have appeared. I remember seeing several such cases in St. Thomas's Hospital, and one or two in other general hospitals when I was assistant medical officer at Bethlem, and was prowling about the general wards to see what one could pick up in one's own line. There one used to see cases which, up to the time of their admission into the hospital, had shown no marks of insanity.

As a rule, however, general paralysis of the insane exhibits very marked symptoms. Perhaps, after all, the best way of studying the varieties is to take it for granted that the *symptoms* are not so

Digitized by Google

characteristic as are the *stages* of the disease; thus, there is a warning or prodromal and threatening stage, followed distinctly by what may be called an acute stage, and by acute I do not necessarily mean that the man is raving; I mean rather that the symptoms have come on precipitately or rapidly, bearing out the old experience that what we call sudden in clinical medicine means generally an old pathology. Just as one's teachers, like Gull and Wilks, used to point out that an acute disease or an acute attack meant that degeneration or change had been going on before—that the acute attacks in the majority of diseases were really attacks on a tissue already decaying or diseased, so one would say that in general paralysis of the insane it is common first to have a threatening or warning, and for the warning to be followed by an acute period, which is then followed by a paretic or indolent stage, and then comes the last or paralytic stage and the accidents associated with it. So we have these three stages. And besides that you have to remember that any one of these stages may be arrested for a time. There are some general paralytics who falsify one's earlier statement that it is an incurable disease, for one has known typical cases of general paralysis proceed into the first stage, and even into the second stage, and then stop; so much so that medico-legal questions of great importance may depend upon these arrests. One remembers a case in which a man had every symptom of general paralysis of the insane. He was seen by several alienists and diagnosed to be a typical case of general paralysis of the insane, and one that was likely to run an acute and rapid course, and therefore he was one who would not be able, probably, to make a will properly. At the end of a few weeks a complete remission took place. He made a will. Afterwards he had a relapse, redeveloped all the symptoms of general paralysis, and died. The will was contested, but it was proved that it was possible to have a complete remission of the disease. Therefore you have to remember that there may be arrest in any one of these stages, during which the person may be able to enter into any contract and to make a will, and, indeed, commit any crime, although one recognises fully that he would not be in all probability looked upon as responsible.

Now to proceed with our subject. What varieties or forms do I intend to point out? First, I will just rapidly go through them. There is (1) the acute, or what may be called the acute delirious kind; (2) the ordinary type; (3) the melancholic type, divided into two sub-divisions, the hypochondriacal and the stuporose; (4) the progressive demented type; and (5), which is less frequently seen and is much more difficult to diagnose, the general paralysis of the double form. Then we have general paralysis chiefly or primarily showing itself in spinal affections, in what has been called (6) spinal general paralysis; in these we have one group which almost always shows (6*a*) the spastic symptoms, with exaggerated reflexes, in another there are (6*b*) the ataxic symptoms. Then (7) for convenience one makes a group containing the female general paralytics. And in that case it is important to remember that there are several peculiarities which almost always characterise general paralysis in women. The duration is rather longer, and the symptoms are almost always associated with exaggerated reflexes, and not with ataxy. Next we have (8) to consider what are from some points of view the most interesting but a rarer form, the adolescent or pubescent or juvenile form of general paralysis. And I still think I am right in saying that not one single case of that kind has yet been observed in which there has not been a history of congenital syphilis. Then comes the (9) senile general paralysis, to which I shall have to refer much more in detail later, because there is much greater difficulty very often in distinguishing whether a man is suffering from simple progressive decay, or whether he is suffering from this specific decay.

One is in the habit of saying the decay of man along his nervous lines may follow the same or similar lines, but at different rates; thus the strong, vigorous, middle-aged man with a syphilitic history decays as a general paralytic in a special way. And one would point out to you that there are certain interesting distinctions that may be drawn. I do not wish to explain. It is better that we should observe and record and let the future explain in many cases. Again one thinks of Sir William Gull. When I propounded some explanation of a pathological nature he said "Savages explain; wise men investigate; and the rain-makers came long before the meeting of meteorologists." He added, "I think at the pre-

Digitized by Google

sent time we will observe." One feels, in relation to general paralysis, that there is a great deal to be observed and is being observed. I am thankful to say that the work which has been done by Dr. Mott in relationship to the Metropolitan Asylums is work that will stand, I believe, for all time and in all countries.

Acute general paralysis of the insane is probably not quite so rare as it appears to be. I will give you a sketch outline. A middle-aged man who has had a great deal of worry and anxiety; who years before had syphilis and had thought comparatively little about it, having been fully treated; leading a free, active, vigorous life—a full life. He lives well and takes stimulants, perhaps not in excess, but freely. He is, his friends say, rather a well-living gouty sort of person. Things go on thus for years; he has a little extra worry and anxiety, and he does not sleep well, and that is an important point. I shall have to point out to you that the general paralytic as a rule, contrasted with the alcoholic, sleeps well; in fact, he very often sleeps extremely well. But the acute case of general paralysis is generally sleepless and restless. Then may come a money loss, an attack of influenza, a feverish cold, so-called, and the man worries in his sleep. His temperature is only 101°, but he is put to bed, and the delirium, instead of passing off, becomes much more like delirium tremens. I have not infrequently, under those conditions, been called upon to see patients, and have been told that they were suffering from the result of alcohol. And certainly they looked very like it; in fact, it is almost impossible to distinguish them. They have hallucinations of sight, they are sleepless, their face is perhaps not so freely perspiring as that of the alcoholic, but distinctly anxious-looking and worn. Very likely you will not be able to examine his pupils, and you may not be able to examine his knee-jerks, and almost certainly you cannot get any further information from him. Instead of opiates or aperients and sedatives or bromides having any effect the man's aspect becomes very grave. His lips get dry; his tongue is parched and cracked; his pulse is running up to 120 or 130, or even more; his food is taken badly, and in some cases if taken is rejected. One sees him again and says, "Well, evidently it is not an alcoholic case, it has none of the aspects of the alcoholic case. It is one of two things: it is either acute delirious mania, or it is acute general paralysis." If it is acute delirious mania the whole thing may pass off with feeding, rest, and careful nursing. If, on the other hand, it is a case of acute general paralysis the man will possibly improve, and you may think that you have done wonders, that your patient has recovered and that he is going to rapid convalescence. You get a view of his pupils and you find that they are unequal. You notice that his speech is affected, and you discover that he has alteration of the reflexes. Before you see him again you are warned that there has been a relapse, that the man is acutely maniacal, not so deliriously maniacal and excited as before, but wild and boisterous, obscene, filthy, and violent. The case runs a rapid course and may be dead in a few weeks. Or the excitement may continue so that the patient passes rapidly into a condition apparently of chronic mania, if I may use the term "chronic" for a disorder that only lasts three or four months. But I have seen patients who were simply ailing as the result, it was supposed, of a febrile attack like influenza, and yet who have been dead in six weeks, with all the physical signs, and all the post-mortem signs too, of general paralysis of the insane. I said just now that there are probably more of these cases than one recognises. There are the cases which were described formerly as cerebritis or brain fever, who not infrequently died, and usually no post-mortems were made; even now you can only say the man was delirious and febrile without any cause as far as you can make out. I believe that the distinction between these cases and the acute cases of general paralysis is a very narrow one indeed; that in fact probably the acutely delirious and maniacal cases depend upon some toxin produced in the brain itself, and the causes of general paralysis depend upon some toxin produced in the nervous system, associated with the original syphilitic trouble. So much for that type. Fortunately it is not very frequent, but it is very fatal.

Next comes the ordinary case of general paralysis of the insane, of which everybody knows pretty well. I do not think you can prophecy from the threatening or the warning symptoms what the type of the disease is going to be. The first disorder very often is a slight depression or malaise; the man is not quite able to do what he did best before. I would put it in that way, that he loses

Digitized by Google

his highest and last accomplishment. The actor ceases to be able to express himself clearly; the man who earned his living by writing no longer writes well; the man who lives by selecting leather is no longer a judge of leather. The painter mixes his colours badly, the draughtsman is faulty in his lines. There is an early degeneration of power in the last accomplishment which he learnt. The loss of this last acquirement is the thing which is most important, and if it occurs in a man of middle age who has lived a fairly free life, who has had syphilis not many years before, be on the look-out at all events for the dangers that may be ahead. If a man with that history has an epileptic fit, never having had any before, be still more on the look-out. If a man of that type becomes unduly emotional and hysterical, I should be very much on my guard. Some of the worst cases of general paralysis I have ever seen have begun with marked hysteria in middle-aged men. The threatening period, as I have said, may last for some time. It may pass off altogether and be forgotten; a temporary loss of power or consciousness, or a fit may occur, or what is still more common, and this marks out the syphilitic relationship, you may get ptosis, an external strabismus, or mydriasis, in a patient who is treated with iodide and mercury, and the whole thing clears up, and nothing more is thought about it. Such a man as I have described has temporary aphasia, temporary hemiplegia, is relieved by mercury and iodide of potassium, or he is sent over to the Continent for general treatment. Those things are forgotten. Again, a return of the ptosis occurs, or more commonly a recurrence of the aphasia may take place, and then after perhaps a second or third recovery the true symptoms of general paralysis show themselves. That is an ordinary type. First of all there may be constant sleepiness, and I think some of you who have served and will serve in the tropics may think you have got cases of sleeping sickness when you see some of these general paralytics. I always wonder what the history of the fat boy in 'Pickwick' was; I think there must have been some mistake about his age, because his trouble was particularly like the early stages of general paralysis; do not forget that the alcoholic degenerate generally sleeps badly. An ordinary case of acute mania generally sleeps badly; but often the general paralytic sleeps unduly well. And even if he is passing into a state of mania he does not complain of sleeplessness as if it were a loss. There is a difference between active sleeplessness—that is, painful sleeplessness—and what you might call buoyant sleeplessness. I see general paralytics who say, "I have discovered how to live now; I only want five hours' sleep a night and I *do* sleep then." You get that sort of thing in general paralysis not infrequently. Then comes the restless irritability, the intolerance of restraint, and then the inundating letters and telegrams. If a patient's friends come to me with a bundle of telegrams in their hands, I am almost sure what has happened. There is restlessness and great activity. No one should condemn a patient as a general paralytic on any one mental symptom; you must have motor symptoms as well. You will hear and know much about the exaltation of the general paralytic; such individuals can understand the folly of another, but cannot at the same time recognise it in himself, like the Bethlem patient who amused the late Dr. Bristowe so much when he was going round Bethlem Hospital. He said, "I am going to build a new Jerusalem." Another general paralytic says, "Get away, I have given you that job." I have had a general paralytic doctor take me round the wards of a hospital for the insane, and point out every general paralytic, although he had every one of the symptoms himself, being totally unable to judge of himself, and yet having all these symptoms. Remember you have on the mental side instability, irritability, and what has been called "facility"—that term is a very good and expressive one; it enables one to treat more general paralytics reasonably than almost anything else. A general paralytic can be induced, like a child, to do many things, differing therein from the alcoholic and very many other similar cases of mania. The alcoholic says, "No"; he puts his back up and says he will not. In the case of the general paralytic, you tickle him with a feather and suggest he should go to Barnum's, or that he should go and see the kennel show, or anything else, and he will agree; he can be easily led. That facility is a very important symptom indeed in this stage. Remember the pupils are almost invariably unequal, sometimes being so much contracted that it is difficult to see whether there is any inequality. They are generally irregular, and almost always immobile. The discs

Digitized by Google

are very rarely affected until the later stages. And though ptosis and external strabismus may be common in the earlier stage, before general paralysis is developed as a true syphilitic trouble, yet when the general paralysis is developed you get very rarely anything like ptosis, and you rarely find there is any drawing of the mouth. You find a want of expression which is very characteristic: the whole of the upper part of the face seems to lose its outlines; and the speech becomes more or less affected. Of course there are certain words which one uses—"Fiscal problems" is not at all a bad one now, but I generally make use of "British Constitution." There are certain combinations of words and letters which will enable you to trip them up as it were. There may be a stage of excitement with great exaltation, with almost universal exaltation. I shall have to point out to you the distinctions between exaltations met with in general paralysis and the exaltations met with in other conditions. But I would here say that in the ordinary type of general paralysis, when there is exaltation it is a universal one. The man is archbishop of the world, he will ride a horse for the Cæsarewitch, and he will dance or he will pray, he will write a book or he will fight a duel, he will govern the world or he will dance in a ballet. You have only to suggest anything and he will agree to do it. Another thing is that the exaltation is nearly always a benevolent one. The paranoiac with grandeur hugs his grandness, and is indignant if you do not at once acknowledge it. But the general paralytic, if he has offered you one million and you ask him for another he will promise it to you, and as many other millions as you like. He is not only facile, but he is benevolent in this stage of exaltation. This stage may last for any length of time, from a week to a year, or even more. Probably the average duration is three or four months of the actual maniacal exaltation. And one would put it in this way: the greater the excitement, the greater the probability — I was going to say unfortunately—of a remission. One is asked to see a person in an asylum who has caused no end of trouble to his wife and friends: he has squandered half his fortune in three or four months, and got into all sorts of moral or immoral troubles; and he is sent to the asylum. Within six weeks he is calm and placid, and his friends, especially his wife, think he is quite himself again, and they would like to have him home. The wilder these people are on admission, the greater is the chance of complete remission. If they are taken out at once when they get into this condition they almost always rapidly relapse. This is what I have called the acute, or active stage.

The next stage is that in which the man gets more or less fat and fatuous. It used to be called by an attendant at the Bethlem Hospital the "piece-bowl" stage. After dinner the pieces not required are put into a big bowl, and if the attendant saw a patient slip along afterwards and help himself to the pieces out of the bowl he at once took it for granted that he was passing into the second, or weak-minded, stage of general paralysis. I have called it the fat and fatuous stage, because in the majority of cases these patients eat, eat, eat, and sleep, and gradually get more and more feeble. Here again the period is a variable one. It may last a few months in an ordinary typical case, or otherwise the patient may remain in the fatuous stage for almost an indefinite time. I have known a patient continue for four or five years, or longer, just eating, drinking, and sleeping, unless some accident takes place. Under nearly all these conditions these patients are liable to accidents, their bones become more brittle, and besides that undoubtedly they bruise very readily; that is to say, they get subcutaneous hæmorrhages and hæmatomata on the ears. That is one of the many evidences of the instability of tissue as well as of the mental instability. From this fat and fatuous stage they generally, but not invariably, pass into the wasting condition. When they begin that they may go on eating, but they are no longer properly nourished. They frequently have diarrhœa; they would develop bedsores if they were not most carefully looked after, and from the stage of paresis they pass into the stage of paralysis, and sometimes die by accident. That is just a sketch outline. Generally there is slight depression as a warning, with possibly sleepiness and possibly some local paralysis due to a syphilitic lesion, some slight motor, sensory, or mental change, comparatively slight, not infrequently recurring; followed by the acute attack of a maniacal nature, with restlessness, buoyancy, exaltation, and benevolence, associated, as I say, with certain physical symptoms—changes in the reflexes, changes in expression, changes in

Digitized by Google

the pupils. There may or may not be fits of various types. There may be simply slight fits, or there may be hysterical attacks, which slightly resemble epileptic fits; or the patients may have most severe epileptiform fits, passing from the fat stage to the paralytic stage, and then death occurs. The whole duration of the disease is said to be about three years. Of course that is on general principles, but if you prophecy on any individual case you are almost sure to be wrong, or, if not, you will gain a greater reputation than you would by curing.

The next type is the hypochondriacal form, and these cases are very misleading, and probably there are more of them than are recognised. A man of middle age, with a syphilitic history, begins to be self-conscious, begins to think there is something generally wrong with his throat or his bowels. He is for ever worrying about his bowels, and he is sure he ought to be taking more purgatives. He is much concerned about this, and he will talk about nothing but his bowels, and then this becomes so far a dominant note that he neglects his living. You will probably think of hypochondriacal melancholia in such a case, but remember also that it may not be that, but it may be a case of general paralysis. I have seen a good many of these cases which have not been detected as cases of general paralysis till there was a want of consistency, and I say that if you get a want of consistency between the physical and the mental symptoms of any case, look out for general paralysis; for instance, a man who appears to be miserable and is always complaining about his bowels, but who is yet getting fat. He may be passing from the acute stage of melancholia into the second, or fat and fatuous, stage. Then the patient may develop hypochondriacal extravagance, like one of the patients, who patted his abdomen with evident satisfaction, saying, "Millions, millions!" And when asked what it was, he said millions of tons of fæces. Another, who was equally satisfied, only rubbing his abdomen and saying it would be such fun when the flood came. He was quite convinced that he was full of water, and so full that when the burst took place there would be need of another ark. Remember, therefore, if a man comes to you with such a history as that which I have mentioned, even though he be melancholic, do not be sure that it is simple melancholia till you have seen how the pupils, the reflexes, and the speech and writing are.

A still more difficult group is that in which simple dementia, simple inability to perform his ordinary functions, comes upon a man, and then he again takes all the food that is given to him. He seems to have no originating power. Put him in front of food and he will go on eating till he almost bursts. He slowly becomes fat and stuporous. And yet there does not seem to be very much which you can mention the matter with him; he is an individual who has a good deal of mental power left. He is dull and apathetic, but if you give him a thing to do he will go on mechanically doing it. That is the type in which you get a heavy, greasy aspect, a very rapid development into the second, or fatty stage. You early get a tendency to bedsores and a tendency to neglect the bowels, so that the stools and the urine are passed involuntarily. I think also such tend to have fits early, which not infrequently end the case quickly. So that a man who rapidly passes from ordinary depression into stupor or dementia is very likely to have fits, which terminate rapidly. On the other hand, he may get some secondary trouble, such as lung trouble, and that may kill him.

Coming now rapidly to the type of paralysis of the double form. Take an example, just one, for I must be brief. A man came to my consulting-room years ago. "Look here, my friend, so-and-so told me to come and see you. It is no good, I have got my luggage on the cab and I am going to the Riviera." He said I am going to do this and that, and "Do you think, doctor, you could come with me? I will pay. We will have a jolly good time, and you shall have everything." He mentioned his brother, and I said "Is your brother going with you?" "No," he said, "he is an old fogey; I cannot be bothered with him." I ascertained that his brother lived in London, and wired, "Stop your brother from going abroad." It was well that he was stopped, for in a short time he was offering marriage to half the barmaids in London, squandering his money hither and thither, dressing in the most extravagant way, and getting into trouble everywhere. He was sent to an asylum, where he was pointed out to me, amongst others, as a typical case of general paralysis. He had all the benevolent exaltation

Digitized by Google

and the physical signs—tremor, unequal pupils, alteration of reflexes, everything,—and in six or eight weeks that man was apparently as well as I was. But he still had the inequality of pupils and a little change in his hand-writing, but practically nothing else. I said, and I think still, that he was a general paralytic, and I do not think he had the saving clause of drink. He was discharged, and he resumed his work as an architect. Between one and two years afterwards I was asked to see him in a most profound state of melancholia, refusing his food and having to be fed artificially, and saying that his soul was lost. I still thought he was a general paralytic. But the people in the asylum said, "You are wrong." He was carefully tended and fed, and once more, three or four months afterwards, he became quite well. He went out. Another year passed, and history repeated itself. A man came to my consulting-room with his luggage on a four-wheeled cab, and said, "I am going abroad this time. I do not care what Ferrier and you say at all." Then he passed through a stage of most acute mania, with all the extravagance and exaltation. Again he got well. Later he had an attack of melancholia; which again passed. After three or four attacks of mania he had fits and died. Those cases are very misleading, if you see them only in the melancholic stage. You may look upon them as simply melancholic. If you see them in the stage of mania and see them get well you think you have made a mistake, and therefore one warns you that there is this double form or type which it is extremely difficult to detect.

In spinal general paralysis there is very little more to be said than that in some cases the spinal cord seems to be affected much more and much sooner than the brain itself. Of course there are two possibilities: that the cord itself may be primarily affected, or it may be that certain motor areas of the brain, associated in one case with the lateral columns and in the other with the posterior columns, are chiefly affected. But at all events, take a good example. A man is brought to see one with marked ataxy; everything points to ataxy, from his boots to his gait. The pupils are contracted. One gets no good view of his discs; there is very little beyond the ataxy to see. But they say he is losing his mental power. I shall have to refer to cases of locomotor ataxy pure and simple, in which also there may be mental symptoms, but I am not referring to those now. These are cases in which ataxic symptoms may exist, and may last for several years, and then there comes an explosion. I have known one case at all events with alternation. When the man was very ataxic his mind was very clear, and when the man's mind was very much disordered the reverse took place, and he was walking well. I remember him so well because in one of his phases he used to assist, when I was playing racquets at Bethlem, in picking up the balls. When he became ataxic he did not recognise the change, and used to fall over when trying to stoop with his usual celerity. But you may get certain cases in which ataxic symptoms precede others, in which everything points to locomotor ataxy and you have no suspicion of general paralysis of the insane, and yet a year or two afterwards that disease may develop. General paralysis of the insane is almost invariably associated with some change in the knee-jerks. There may be exaggeration of these reflexes on both sides; there may be either absence or defect on both sides. Or you may have irregularity: there may be exaggeration on one side and loss or diminution on the other. But remember that there are cases in which either the spastic condition or the ataxic condition may precede ordinary general paralysis. And the only other thing I would say in reference to this in passing is that any of these cord states may be associated with any form of general paralysis. You may have locomotor-ataxic symptoms preceding an attack of general paralysis which ends fatally in a few weeks. You may have it associated with melancholia. It is not uncommon to have melancholia in cases of general paralysis preceded by ataxic symptoms.

In women, I have told you, we get few cases compared with the number in men. Now and then one has seen, perhaps four or five cases, in which both husband and wife have suffered from the disease, and one has had a most typical demonstration of the syphilitic relationship of the disease to general paralysis. One man who had general paralysis, a somewhat distinguished man, had had syphilis many years before he developed general paralysis. A year or two after he developed general paralysis his wife developed the same condition, there having been very marked evidence that syphilis had been communicated. In general paralysis in women you very rarely get ataxic

Digitized by Google

symptoms, and that is of course interesting; just as it is very rare to meet with a woman who has locomotor ataxy, so it is very rare to meet general paralytic women who have any ataxic symptoms. And that is one of the questions which one cannot explain. Mr. Jonathan Hutchinson is inclined to think that that depends on the sexual relationship of the two, that in men sexual pleasures tell much more gravely than they do in women; and therefore he would be inclined to look upon locomotor ataxy as much more frequently aggravated, if not started, by sexual excess, and likewise he thinks that the locomotor-ataxic symptoms in general paralysis in men are likely to be chiefly associated with sexual indulgence.

Adolescent general paralysis may occur at any age, possibly in infancy. There seem to be some of the cases of convulsions in children that are related undoubtedly to the syphilitic inheritance, and we have, as I say, a group of cases of general paralysis of the insane in which you have got a good many peculiarities. You have rather to judge here from the distinct syphilitic inheritance, the exaggerated reflexes almost invariably, the pupillary inequality, and the great difficulty of speech. There is generally pretty rapid and progressive dementia. And I should warn you in this way: If you have a case of a young fellow or lad brought to you, and you think—well, this is undoubtedly a case of disseminated sclerosis, be on the look-out. I have seen a very considerable number of cases that have been diagnosed as cases of disseminated sclerosis that have turned out to be juvenile general paralysis. You get in these cases sometimes maniacal excitement, but I think it is rare. As a rule, you get great irritability, great restlessness, with a rapid passage from the first stage to the second, when you get the most troublesome, irritable, dirty patients that you can imagine, passing more or less rapidly into the paralytic stage. And then they die, sometimes with a fit, sometimes without. I am afraid I shall have to defer the full consideration of senile general paralysis to the next lecture, when I come to the diagnosis between general paralysis and the other forms of nervous disorder. One would say that there is often great difficulty in distinguishing between the decay in old men; that is to say, between the comparatively normal decay of senility and the decay dependent upon some syphilis as well as age.

Henrietta Street, W.;
March 7th, 1904.

PROLIFERATING PAPILLOMATOUS OVARIAN CYSTS.

By E. STANMORE BISHOP, F.R.C.S.Eng.,
Hon. Surgeon, Ancoats' Hospital, Manchester;
Vice-President British Gynæcological Society;
Ex-President Clinical Society, Manchester.

GENTLEMEN,—Although ordinary ovarian cystoma is of frequent occurrence, its course and treatment well understood, and the results of such treatment by removal so uniformly good that it has ceased to attract much attention, occasionally unusual cases occur, which may repay more careful consideration. Ovarian cysts are uni- or multilocular; but, more scientifically, they may be divided into those arising from the germinating cell-layer, the oöphoron; those arising from the paroöphoron, the fibrous portion below, which contains remnants of the mesonephros; and lastly, those originating in the parovarium, which contains remnants of the Wolffian body.

From all these cysts may arise, which differ according to their point of origin; thus, from the germinal layer arise the simple cysts which are enlarged ovarian follicles: adenomata—the usual multilocular cyst—and dermoids. These project into the cavity of the peritoneum, and the Fallopian tube lies below them.

From the paroöphoron, or rather from the remnants of the mesonephros contained therein, arise uni- or multi-locular cysts which contain papillomata. These project into the cavity of the peritoneum, and the Fallopian tube lies beside them.

From the parovarium, or rather from the remnants of the Wolffian body contained therein, arise unilocular cysts which do not contain papillomata, but may contain low, flat-topped warts. These lie below the peritoneum between the layers of the mesosalpinx and broad ligament, and the Fallopian tube, sometimes greatly elongated, is stretched over and above them.

I wish to-day to direct your attention specially to cases which come under the second variety, viz. papillomatous cysts springing from the paroöphoron, and to bring before you the records of two cases occurring lately in my practice, illustrating their history and appearance.

Papillomata also are seen in the Fallopian tube, and in one of these cases, the first, this was seen at

Digitized by Google

the second operation. Bland-Sutton, however, after histological examination of such growths in this situation, considers them to be true adenomata, though growing in a dendritic form.

The first case affords a somewhat unusual opportunity of observation for a comparatively long time of the course of events after operation.

In November, 1898, I was requested by my friend, Dr. Knapton, of Bowdon, to see with him a very stout woman who complained of abdominal enlargement with great aching over the sacral region and around the hips. Dr. Knapton had attended her for some weeks, and had at first detected a lump of the size of a small football on the left side of the abdomen, not accompanied by any free fluid in the abdomen. Not long after as she was going upstairs she became giddy and fell over the banisters into the hall of her house. As she was a very heavy woman, the fall was a serious one, and Dr. Knapton was sent for. On examining her he found only slight injuries, but the abdominal lump had disappeared, whilst there was now evidently free fluid in the peritoneal cavity. The size of the abdomen decreased for a few days, and then rapidly increased so as to interfere with her respiration. Dr. Knapton accordingly tapped her on November 21st, 1898, but found that the fluid present was of a thick glutinous character and ran out with some difficulty. Three and a half gallons of this material escaped, and of course the abdomen became flat again; but it very rapidly refilled, and six days afterwards when I saw her she was nearly as full as before. At that time certainly no mass could be felt, and the contents of the abdomen were perfectly free in the cavity.

She was forty years of age, married, and had had three healthy children, but no miscarriages. Her youngest child was fifteen years old, and she had been a widow for fourteen years. Menstruation began early, but she did not know how old she was at the time. It had always been regular, except during her pregnancies and lactations, with some pre-menstrual pain commencing one or two days before, and passing off as the period became established. This pain had never been worse on one side than on the other; since the fall blood had appeared every fortnight. She was very fat, and had marked enteroptosis. She could not say exactly when she first noticed the abdominal swelling, but it was certainly less than six months. Her daughter had noticed that for about that time she had been in the habit of placing her hand over the left iliac region, as if for support. For some time, and until the tapping was done, she had had difficulty in urination, not being able to pass much at a time. Since paracentesis she had passed urine freely. Her eyes were prominent and bloodshot, but this had been the case for some years. There was no detectable cardiac, renal, or hepatic disease.

The character of the fluid obtained on tapping showed clearly, apart from the history of a previous mass, that what was now present was not ascites. It was believed that a multilocular glandular ovarian cyst had been present, and that this had given way. The only objection to this was the fact that after the tapping no remains of the cyst could be felt. Glandular multilocular cysts have almost always some portions which are solid, and which therefore remain and are palpable after the more cystic portions are emptied of their contents; but such portions may be but small—this cyst had evidently not been growing for any great length of time,—and they might have been overlooked. At the time when we both examined her there was too great a return of the effusion for any certainty on this point. The rapidity of return and the large amount of it pointed to papilloma, but against this there was the fact first noted, that no free fluid was present when the cyst was intact, that no blood had been present in the fluid evacuated, and it was felt that it was possible that some hydro-peritoneum might have been caused by the traumatism.

As it was evident that something more than a repetition of the paracentesis was required, it was decided that the abdomen should be opened, and accordingly, on the 30th of November, 1898, this was done. There was an immediate gush of amber-coloured glutinous fluid on opening the peritoneum. The omentum which protruded was thickened and lumpy. On passing the hand down towards the uterus it encountered a soft, pulpy, slimy mass, which, on being brought up to the level of the wound, resembled the villous interior of inflamed small intestine. When drawn out it was seen to be attached by a narrow pedicle to the left broad ligament, which appeared to be connected with the centre of the interior of a cyst. The cyst, after bursting, had apparently turned inside out, so that what was first felt and seen was its true interior, covered by papillomatous growth. By the side of

Digitized by Google

the mass lay an apparently normal Fallopian tube. The pedicle was tied and dropped. No other growth could be felt. The opposite—right—ovary and tube were examined, and nothing abnormal being found in them, were not removed. Still large quantities of pulpy fluid poured out, coming apparently from all parts of the abdominal cavity.

At this time the folly of using drainage-tubes in the peritoneal cavity was not recognised, and two large glass tubes were placed side by side in Douglas' pouch, their open upper ends projecting from the lower end of the abdominal opening, the rest of which was closed by tier suture. The abdominal cavity was then irrigated through these until the fluid returned clear. As she recovered consciousness she began to retch, and large quantities of fluid escaped again, soaking through the dressings and covering the table.

At night, ten hours after, large quantities of glutinous fluid were reported as having flowed out, soaking the bed and causing great difficulty in the task of keeping her dry. The next day one tube was removed, and on the following the other; and she made a good recovery, leaving for home on the 27th of December, about a month after the operation.

For some months she was perfectly well, but in August of the ensuing year she again noticed an increase in size. In September the swelling increased more rapidly, and she began to have pain on the right side. In October this pain became much more severe, and she suffered from loss of sleep. During the last fortnight before examination she began to lose weight.

On October 31st, 1899, some free fluid was found; along with this a bilobed mass was felt in the hypogastrium, extending from the pelvis upwards for some inches above the umbilicus on both sides. The abdominal wall was tender on pressure over the left iliac region—not skin tenderness. She was unable to lie on either side and was rather breathless. The uterus was mobile, the os split on both sides to the vaginal vault; there was some follicular degeneration of the cervix, but the surface was smooth and there was no cheesy sensation on using the curette. Some bleeding, but not more than the use of the curette would account for, followed.

The abdomen was again opened on November 1st, 1899, and as before, on opening the peritoneum there was an abundant gush of yellow glutinous fluid. A mass the size of both fists was found springing from the edge of the right broad ligament. Its pedicle occupied the site of the Fallopian tube. By its side, and external to it, was the unchanged right ovary—unchanged except for the presence of a very small cyst. Behind the uterus, in Douglas' pouch, was another but smaller semigelatinous mass attached by a broad base to its posterior surface. The pelvic peritoneum generally was injected and reddened, and several smaller papillomatous growths were scattered over its surface. A coil of small intestine which had occupied Douglas' pouch was covered by a finely villous surface. The mass and ovary were removed, as well as as much of the secondary masses as could be easily peeled off, but a good deal of this had to be left. By this time the advantages of reconstructing a complete peritoneum had been recognised, and greater confidence in its powers of absorption created both by greater experience and by Clark's work in reference to this subject; therefore, after thorough washing out with saline solution, the abdomen was entirely closed without drainage by tier suture and sealed with celloidin.

She again made a rapid recovery, and was not seen again until January, 1902—two years later. At that time she returned complaining of vaginal discharges mixed with blood, and on examination there was evident carcinoma of the cervix. This was confined to the cervix, the uterus being quite mobile; the vaginal mucous membrane and broad ligaments were quite free from growth.

On February 2nd, 1902, vaginal hysterectomy was carried out without difficulty. On removal of the uterus no fluid followed, the peritoneum was normal as far as could be seen, and it was specially noted that all trace of the secondary patch of papilloma which had been left on the posterior wall of the uterus had disappeared, leaving no trace of its previous presence. Once more recovery was uneventful, and she returned home on March 20th, 1902, in apparently perfect health.

She is now, December, 1903, in very good health and spirits.

The second case had by no means such an eventful history, but such as it is, it serves to complement the other.

Miss G—, æt. 40, was sent to me by Dr.

Digitized by Google

Partington, of Tunstall, in August, 1903. She was unmarried. Her menstrual history began at fifteen years, and she had always been regular ever since. She had always had a free flow, lasting for about five days. At first there used to be some pain at the commencement, but latterly this had disapppeared, and the amount lost seemed to be becoming less. She described herself as feeling perfectly well in June last, but soon afterwards she was conscious of a feeling of weight in the abdomen, especially if she walked any distance.

When she was seventeen years old she had an attack of breathlessness, and apparently suffered from anæmia. A similar attack occurred once again, when she was twenty-two.

She first noticed an increase in the size of the abdomen about eight weeks previous to examination, and since that time defæcation had been at once more difficult and the need when it occurred more urgent. Urination had always been perfectly easy.

At the time of examination the abdomen was markedly distended; the abdominal veins were, however, not enlarged. There was no free fluid in the abdomen. On the left side mainly, but extending across the right iliac region, was a large rounded tumour, of unequal consistence, movable laterally, dull on percussion—the dulness extending upwards from the pubes and left Poupart's ligament,—but surrounded by clear sound above and on the right side. Nothing could be felt per rectum, the cyst not extending into the true pelvis, and the uterus was virgin and in the normal position. Its movements were not conveyed apparently to the tumour above. A diagnosis of multilocular ovarian tumour was made.

On September 16th, 1903, the patient being in the Trendelenberg position, median cœliotomy was performed. A large cyst presented, which was very adherent above to the omentum, below and in front to the left broad ligament, the uterus, and even to the right broad ligament. This was tapped and lifted out as far as possible; it was not possible to bring it far, owing to the adhesions. After the omentum the left ovarian artery was tied, and separation began from this side. Soon after a cavity was opened, from which brown thick material oozed out. Fearing that the sigmoid had been injured, as the material resembled fæces in everything but smell, this opening was packed around by sponges, as much of the exuded material wiped away as possible, and the opening closed. The cavity was in a structure which lay in an antero-posterior direction across the outer extremity of the broad ligament, therefore somewhat in the position which the sigmoid might have assumed if much tied down by adhesions. Turning to the opposite side, the Fallopian tube and ovary on the right side were left, a division being made on the uterine side of the right broad ligament. Some portion of the uterus, about one inch of the fundus and body, was necessarily cut across in order to remove the whole of the growth, and, working in this way backwards to the left side, the entire tumour was at last freed and lifted out. Several isolated papillomatous growths were found in Douglas' pouch and scattered over the pelvic peritoneum. One, the size of a small orange, was seen in the omentum and just above the free extremity of it. The latter was tied off, and as much as possible of the former was peeled away. Now that the main mass was gone it was possible to follow the course of the sigmoid, which lay behind and free from the structure which had simulated it. This was found to be closed at both ends, and was dissected off the broad ligament. It proved to be a dilated Fallopian tube, convoluted as to its inner and outer thirds, and greatly hypertrophied in the middle third. The cavity there formed was full of the same brown thick material, which was evidently old blood-clot. No papillomatous growth was, however, found in this. Its lining membrane was quite free from growth, was lying in longitudinal folds, and surrounded by a well-developed muscular coat. Even after removal, its resemblance to a piece of intestine was very great.

The abdomen was closed by tier suture, the fascia being united by a modification of Milton's method.

Recovery was somewhat slow, and for the first week post-operative insanity was feared, as the patient talked incessantly, and it was difficult to obtain sleep, but ultimately she perfectly recovered, and returned home on October 30th, six weeks after the operation.

Apart from the comparative rarity of these cases they illustrate certain points, to which I ask your attention.

First there is the rapidity of abdominal enlarge-

Digitized by Google

ment in these cases. Ordinary unilocular and multilocular ovarian cysts are rather slow in growth. As nowadays they are usually removed as soon as they are diagnosed, we must turn to the accounts given by authors who wrote whilst yet it was believed that their removal was accompanied by great risk, in order to obtain a view of their natural evolution. Dr. West,* who wrote in 1858, and who objected to ovariotomy on account of its great mortality at the time, giving a list of 56 deaths out of 118 cases which he had collected, speaks of a case which, after eight years' watching, continued of the same dimensions during the whole of that time, being rather smaller than a fœtal head. "As a general rule," he says, "the enlargement goes on not continuously, but by fits and starts; there are yet almost always seasons during which it remains stationary. Simple cysts now and then remain stationary for many years, life being not at all shortened, scarcely even embittered by their presence." He quotes Stafford Lee's statistics of 123 cases, of which 38 lasted one year, 25 two years, 17 three years, 10 four years, 3 five years, 14 five to ten years, 1 even fifty years; 73·9 per cent. terminating fatally within five years. Spencer Wells,† writing in 1882, estimated the average duration of life with ovarian disease at four years.

In the first case, whilst, as usual, there was a difficulty in fixing the date of onset in the first instance, the patient was positive that the condition had not been present for more than six months; and in the second, her past experience rendering her somewhat acute in recognising symptoms, she first noticed increase in size in August, only three months before the second operation, when the abdominal cavity was full enough to cause apnœa, so that the rate of increase was much more rapid than is usual with the ordinary ovarian cyst. Such rapid enlargement is not always due to the actual size of the tumour itself. It is often owing to the hydro-peritoneum which accompanies, as a rule, these papillomatous growths.

Beyond this quicker growth and the usually concomitant presence of free fluid in the peritoneal cavity, both of which are not invariable, no symptoms have been noticed which will differentiate these cysts before operation from others which spring from the oöphoron. Their presence must therefore always, for the present at least, be a matter of doubt until cœliotomy has been done. This is, however, a matter of no moment, since the treatment and prognosis after operation are the same.

Secondly, the appearances seen on opening the peritoneum. Almost always a large amount of amber-coloured mucoid fluid escapes; even when this has emptied itself sufficiently we do not see the bare cyst with its clean mother-of-pearl-like sheen, which is so characteristic of cysts developed from the oöphoron. If a cyst is seen at all its colour is dull grey with small ridges or tufts of red cocks-comb-like growth springing from it. The cyst is usually adherent to the parts around, generally to the omentum, sometimes to intestine. The parietal and visceral peritoneum is irritated, reddened, and villous-looking in patches. Secondary growths are fairly frequent; but these are always limited to the peritoneum, and chiefly to the lower half. It is not often that a mass is found in the omentum as in the second case.

But these cysts are very prone to yield, and then their appearance is most unusual. They do not simply open at one point, as is the case in some other cysts. I have seen a simple round hole the size of a shilling in a multilocular cyst which looked as though it had been punched out, whilst the rest of the tumour was unchanged. These cysts, on the contrary, open out, and often turn inside out, so that we see the interior of them and not the exterior wall. As this, the interior, is covered with dark brownish dendritic growth, it may not be at first visible in the depth of the pelvis. If the hand is passed down to find the uterus it comes in contact with a soft slimy mass, which feels like a jelly-fish, but on feeling around this the fingers detect the edge, and on lifting this a narrow pedicle can be felt beneath, connecting the main mass with one or other of the broad ligaments.

Thirdly, there is the evidence as to the disappearance of secondary growths after the removal of the primary cyst. In the first case, at the second operation a large amount of villous growth was left behind; some of the intestine was obviously infected, and especially a considerable amount was left attached to the posterior uterine surface in Douglas' pouch, yet at the removal of this uterus for carcinoma two years later no trace of papillomatous growth was seen, the abdominal cavity

* 'Diseases of Women,' part ii, p. 125, etc.

† 'Ovarian and Uterine Tumours,' 1882.

Digitized by Google

contained no fluid, and the posterior uterine wall was perfectly smooth and covered with normal peritoneum. This was a striking proof of the power of the peritoneum to remove secondary deposits, and a complete justification of the advice not to be unduly careful in detaching them at the time of operation.

Lastly, as to treatment. The cyst is freed from any adhesions, lifted and brought outside the abdominal wall, its pedicle is tied and divided above the ligature; then the peritoneum is drawn over the raw extremity of the pedicle by fine catgut sutures. All secondary growths of any size are removed by peeling off the peritoneum, but smaller masses are left untouched. Above all, no attempt is to be made to remove villous surfaces on the intestine. Experience has shown that all these will rapidly disappear when once the cause, the original tumour, is removed. The peritoneum is cleansed as far as can be done without rubbing or any interference with its endothelial surface. Some irrigation with saline solution may be done, but this is not imperative. It is in all cases advisable to err on the side of doing too little rather than too much. Clots of blood should, however, always be removed. Then comes the question as to whether the abdomen should be at once and completely closed, or whether drainage should be employed. At the commencement of the first case, five years ago, most gynæcologists still believed in the advisability of draining the peritoneum in certain cases, if not in all. In the first case a large amount of semi-glutinous material was present, which indeed flooded our dressings, bed, and patient for two days after the operation. At that time it was deemed unwise to close this up and trust to the peritoneum alone, since it had already become so much altered by the character of the growth in it and the contact of the fluid for fourteen or fifteen days, for its absorption. The abdomen was therefore drained by a supra-pubic glass tube. But for some time surgeons had felt that these drainage-tubes were great sources of danger; if they were outlets for material from within, they were also necessarily openings for the entrance of material from without, above all for the entrance of micro-organisms. It was also extremely difficult to determine when they should be removed. If removed too soon they might have to be replaced, a particularly difficult and dangerous thing to do, since the small intestine at once took up the position they had occupied; if removed too late the fluid withdrawn from them became once more bloody and sometimes even purulent; they required constant movement, otherwise their openings became occluded by omentum or lymph covering the tissues around, which was forced into these openings by the intra-abdominal pressure, obstructing further flow and often locking them in such a way as to render their removal a work of great difficulty and risk. Occasionally, as in one case of my own, they pressed upon a coil of intestine which had slipped beneath them during these constant movements, and produced ulceration of the gut with consequent fæcal fistula or extravasation. It was a great relief to be able definitely to abandon their use entirely, which became possible after Clark's* epoch-making paper in 1897. Clark showed clearly that these drain-tubes did not really drain; that very quickly after their insertion the abdominal cavity around was shut off from them by a tube of lymph, which entirely surrounded them; that collections of pus might exist within a few centimetres of these tubes, unrelieved and undrained by them; that such collections had been found post-mortem; that gauze placed in the drainage-tubes became infected with pyogenic micro-organisms from *without;* and that, and this was the most important of all, the peritoneum itself was capable of disposing far more safely and completely of all sources of danger if certain simple precautions, such as the removal of visible clots, etc., were complied with. I believe that this is the last case in which I have attempted to drain this cavity by artificial means, and in all the long list of cœliotomies, both abdominal and vaginal, I have operated upon since I have always, no matter what the condition, aimed at what I now consider to be a primary necessity for success—that is, to reproduce at once and entirely the *closed* peritoneal sac. The difference in the mortality of my cases has been most striking, and the almost total absence of sequent peritonitis most gratifying and convincing. Besides this there has disappeared the possibility of ventral hernia, which almost always sooner or later occurred at the weak point in the abdominal wall left by the drainage-tube.

And, *à propos* of ventral hernia occurring after these operations, it has, I think, been thoroughly

* 'Amer. Journ. Obstet,' 1897, vol. i, p. 482.

Digitized by Google

demonstrated that this is due to imperfect union of the fascial plane, which is acted upon by the contractions of the lateral muscles, whose tendon it is. If this tendinous expansion is thoroughly united these contractions are neutralised, those of one side by those of the other. If it is not, and fat intrudes between its cut edges at any point, a weak spot is left, which these contractions constantly tend to increase. Such a spot may not be recognised until the patient has been walking about for some time, but sooner or later it will demonstrate its presence. It is obvious that no external binder will neutralise this muscular pull; and therefore through and through suturing, which leaves the tendinous union to chance, must be abandoned for tier suture. Tier suture, however, has one great drawback. Sufficiently strong material must be used in order to resist the muscular pull. If this is buried it will not be readily absorbed when its period of usefulness is over, and may later give rise to a buried stitch abscess, the result of which is equally fatal to success. Milton, of Cairo, has quite lately shown how such sutures may be placed so that when the union is sufficiently firm they may be removed. I have adopted a modification of his method which has so far given me perfect results. After suture of the peritoneum with cat-gut a long silver wire is placed over one side of the fascial opening. With an eyed needle a double strand of silkworm gut is passed through the skin and fascia of the opposite side from above downwards, then through the fascia opposing from below upwards. The needle is removed, leaving a loop through which one end of the wire is passed. Beginning just beyond one end of the wound, loops are passed in this way at intervals of half an inch, the wire being threaded through all. When all are in position the wire is held strongly at each end to prevent kinking whilst the free ends of the silkworm-gut sutures are tied over a thick strip of gauze placed lengthways along the side of the wound and between the two threads of each loop. When all are tied the free ends of the wire are brought up in the wound and tied to the extreme suture at either end. The skin is then united by separate sutures. After five to six weeks the wire is divided at one end close to the skin and drawn out from the other end. This sets all the loops free, and they can be drawn out separately, leaving the fascia united and without any buried foreign body in contact with them. This certainly seems a great advance upon previous methods.

Manchester;
February 29th, 1904.

THE RESULTS OF LORENZ'S TREATMENT OF CONGENITAL DISPLACEMENT OF THE HIPS.

SIR,—My friend, Dr. V. Gibney, of New York, in a letter to me dated December 24th, 1903, in answer to my inquiry as to the results of the operations for congenital displacement of the hips performed in America upon the plan of Lorenz, sends me the following report, which may be of interest to English surgeons.

I am, yours faithfully,
E. NOBLE SMITH.

Copy of Paragraph of the Surgeon-in-Chief's Forthcoming Annual Report to the Board of Managers of the Hospital for Ruptured and Crippled, New York.

"Referring to the records, I find that thirty-three patients have been operated upon during the past year by the bloodless method. In order not to confuse you, I use the term patient advisedly, and not cases. If the latter term were employed, every patient with a dislocation of both hips would represent two cases. Of the thirty-three, *eighteen* were under seven years of age, *eleven* between eight and ten, *four* between ten and twelve. Three have already been cured. This means that the plaster of Paris has been removed, and that the head of the bone remains where it was placed, and that the patients are walking without an appreciable limp, the functions of the joint being perfect. Three others ended in good results; that is, the plaster is removed, the functions of the hip are good, but the limb is not quite as long as the other by reason of lack of development of the bones which enter into the formation of the joint. Eight others, at the present writing, are in what is known as a favourable condition; that is, the prospects of cure are good—the dislocation was reduced at the operation; at the end of six months, on the removal of the first plaster-of-Paris dressing, the good position secured remained, but plaster of Paris, with the limb straight and parallel with its fellow, is still worn, in order to induce more stability. Another

Digitized by Google

eight were improved. This means that it was impossible to get the head of the bone into an anatomically perfect position, but that the position was improved by what is called 'anterior reposition.' Three of these no longer wear plaster of Paris; five are still in this fixed dressing, but with the limb parallel with its fellow. All are walking about, and the prospects are very fair for greater usefulness of the limb. Four are put down as failures. That is, by reason of the age it was not possible to reduce the dislocation without incurring unwarrantable risks. It is believed, however, that even these 'failures' will result in improved locomotion as the children grow older. Five are still in the first plaster-of-Paris dressing, the dislocation having been reduced. It is not yet time to change the position of the limb. In one of these failures there was produced a paralysis of the muscles of the leg, but the power is returning, and the outlook is very good for complete restoration. One death occurred, in a girl æt. 12, a case which we undertook with great reluctance."

Remarks by Mr. Noble Smith.

The above report contrasts very favourably with some previous records of the results of manipulative treatment.

Having had some experience in dealing with these cases by Lorenz's method, I would suggest that, in order to avoid the serious accidents which have been described by some surgeons, the following rules should be observed:

First: That the operation by manipulations should be only practised in favourable cases.

Second: That in all cases there should be a preliminary treatment by exercises and stretching.

Third: That in attempting reduction there should be avoidance of severe force.

Fourth: The subcutaneous division of adductors together with rupture of the deeper fibres, should be substituted for Lorenz's method of hand-sawing.

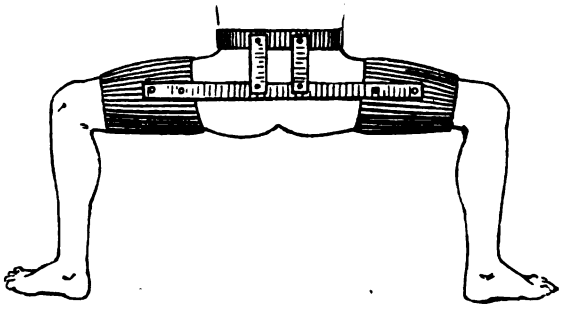
Fifth: That a splint be used instead of plaster-of-Paris bandages.

I will enlarge upon these suggestions as follows:

First: The age of the patient no doubt has a great influence as regards a favourable prognosis, but I do not think we can yet lay down any absolute rule upon this point. Some cases of ten or twelve years old have been more easily reduced than others of four and five. If the false joints are firm and there is very little telescopic action, there will probably be difficulty in reduction, and at the same time such cases will do very well without any operation.

Second: The preliminary exercises and stretching and extension should be carried out sufficiently long to enable the limb of the affected side to be flexed until the foot lies by the side of the patient's head, and these exercises should chiefly be directed towards abduction.

Third: It is difficult to lay down a rule as to the degree of force to be used, but the force ought to be entirely within the control of the surgeon conducting the operation. A case is reported in which five assistants and the operating surgeon were all exercising their strength at the same time on the patient, a child æt. 4.



Noble Smith's splint for fixation of the thighs after reduction of congenital displacement of hips (seen from the back).

Fourth: The possibility of division of the muscles by hand-pressure is very interesting and remarkable, but, except in very young children, it leads to a dangerous amount of ecchymosis and damage to the tissues. Subcutaneous section of the chief mass of contracted muscles, followed by forced separation of the deeper fibres, will be perfectly effective and devoid of danger; or the muscles may be divided through an open wound.

Fifth: A simple iron splint passing across the buttocks will hold the limbs in the required position, will allow observation of the condition of the parts, and will permit of re-adjustment if desirable. Moreover, the splint can be applied in a few minutes, whereas the use of the plaster casing necessitates the patient being nearly an hour longer under the anæsthetic.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 594. WEDNESDAY, MARCH 16, 1904. Vol. XXIII. No. 22.

CONTENTS.

PAGE

*On Predisposing and Pre-cancerous Conditions of the Breast. By HENRY T. BUTLIN, D.C.L., F.R.C.S. 337

*Four Lectures on Normal and Abnormal Conditions of the Blood. By EDMUND CAUTLEY, M.D., F.R.C.P. LECTURE I 343

*With Dr. Charles W. Chapman's Post-Graduate Class at the National Hospital for Diseases of the Heart ... 349

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

ON PREDISPOSING AND PRE-CANCEROUS CONDITIONS OF THE BREAST.*

By HENRY T. BUTLIN, D.C.L., F.R.C.S.,

Consulting Surgeon and Lecturer on Clinical Surgery to St. Bartholomew's Hospital.

GENTLEMEN,—I do not propose in this lecture to consider the conditions of the nipple and areola which predispose to cancer, but only those which actually occur in the substance of the mammary gland. Of causes which predispose to or which precede cancer of the breast there are, I believe, three:—(1) Innocent tumours; (2) duct-cysts; (3) chronic mastitis, with or without the presence of cysts.

Innocent tumours and cancer.—I do not know whether the first is really a predisposing cause of cancer. I cannot remember ever myself to have seen an innocent tumour transformed into a malignant tumour, but there are cases in which there can be no reasonable doubt that the transformation has occurred, and I have seen the microscopic sections of more than one such case at the meetings of the Pathological Society. The preceding growths were fibro-adenomata, and the malignant disease was in most, if not in all, cases carcinoma. There may have been no greater liability of the fibro-adenoma to become cancerous than of any other part of the same breast, and it is not possible to charge the innocent growth with having attracted the malignant growth to that breast or to that patient; so that it is not possible to say with certainty that innocent growths in the breast predispose to the occurrence of malignant disease. Nevertheless there are sufficient cases on record to make it very undesirable to leave such tumours indefinitely on the ground that they are

* Being the substance of a clinical lecture delivered at St. Bartholomew's Hospital.

Digitized by Google

harmless, and I would always advise their removal within a certain definite period of time.

Duct-cysts and cancer.—In at least two instances I have seen the development of cancer in connection with duct-cysts, and the first of these sufficed to occasion a change in my attitude towards these and other conditions which may predispose to cancer.

A lady, unmarried, and æt. 35, consulted me in the summer of 1895 on account of a small cyst of the lower part of the breast. There was no doubt of its nature, because clear fluid could be squeezed out of it through the galactophorous duct which corresponded with it. It had been there a long time, and did not appear to have undergone any change since it had been noticed. I told her what it was and advised her, if it gave her trouble or if it increased in size, to have it taken out. I never saw her or heard of her until the late autumn of 1899, four and a half years later, when she came to tell me that the discharge had become more abundant and was generally stained with blood. To my great distress I found that the site of the former cyst was occupied by a solid tumour, which proved on removal to be a duct-carcinoma. Recurrence followed the free removal of the disease and necessitated the amputation of the breast and removal of the contents of the axilla in 1900. Since then there has been no recurrence.

The second case occurred quite recently in the person of a lady who had had the breast "watched" over a period of many years on account of a small cyst close to the nipple, which discharged through one of the galactophorous ducts. Recently, although the discharge had continued and remained much the same in appearance, she had experienced uncomfortable sensations in the tumour, and that led to a consultation and to removal of the breast for what was found to be a duct-carcinoma of limited extent.

Of these duct-cysts, then, it may certainly be said that they are a source of danger, and that they should be removed while they are small, when an operation of trivial extent may suffice to rid the patient of a present annoyance and of a future dread.

Chronic mastitis and cancer.—And now I come to what is probably by far the most common predisposing cause of cancer of the breast—chronic mastitis, with or without the presence of obvious cysts. I speak of "obvious cysts" because there are many lumps formed as a result of chronic mastitis which appear to be quite solid, but which, when they are cut into, are found to contain cysts. And a microscopic examination will often discover very tiny cysts which would quite have escaped observation without the microscope. So frequent is the association of cysts with indurations due to chronic mastitis, and so easy is it (with the microscope) to discover the evidence of inflammation in the immediate neighbourhood of single and of multiple cysts, and so frequently are unsuspected tiny cysts found in the neighbourhood of a large single cyst, while the intervening tissues are slightly indurated and are the seat of microscopic inflammatory changes, that I have serious doubts whether glandular cysts are not in every instance produced by inflammation. Chronic inflammation of the breast is generally spoken of as if it belonged to and were confined to the connective tissue of the breast. But it is very rarely the case that the glandular epithelium is not involved, and sometimes the stress of the inflammation seems to have fallen on the glandular apparatus. In these photographs the changes which occur in the connective tissue and in the glandular elements, with the earliest development of cysts, are admirably shown. They are taken from disease which I have myself removed. And they are exceedingly well photographed in an excellent paper by Mr. Paul (of Liverpool) in the 'Transactions of the Pathological Society of London' (vol. lii, p. 30, 1901).

The tangible signs, then—for they are often not visible,—of chronic mastitis are either indurations or cysts or a combination of the two. There may be a single cyst, around which there is slight and scarcely perceptible induration. Or there may be more than one perceptible cyst, and between the cysts the tissues of the breast are hardened. Or there may be an ill-defined lump, in which no fluid can be perceived. If it be cut into the lump may be solid, but often it consists of several small cysts in indurated tissues, but the cysts are too small to permit the feel of fluid.

Diagnosis of chronic mastitis.—I can give no accurate instructions for the *diagnosis* of chronic mastitis, for it is often quite impossible to be sure of the nature of the disease. Thus, although it is much more common in women at and after the climacteric period, it may yet occur at any period of adult age,

Digitized by Google

and may occur in girls between puberty and adult age, although I have scarcely ever seen a case. It

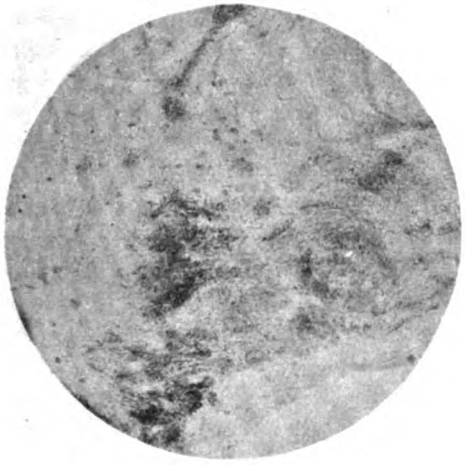


FIG. 1.—From an induration due to chronic mastitis in the breast of a lady fifty years of age. It shows several foci of inflammation. Each focus contains a lobule of the mammary gland, but the glandular structure is almost obscured by the inflammation.

is not in the least limited in its occurrence to the puerperal period, nor does it appear to be so frequent then as later. But it does occur with tolerable frequency about the time of the cessation of the catamenia. Hence the frequent occurrence of cysts of the breast at that period of life; hence the name of involution cysts of the breast, since the cysts are regarded as in some manner connected with the involution changes which are natural then. And hence a very natural deduction, that the formation of cysts is a natural part of a natural process, and that they are therefore quite harmless, to be regarded as no worse than little bags of fluid in the breast.

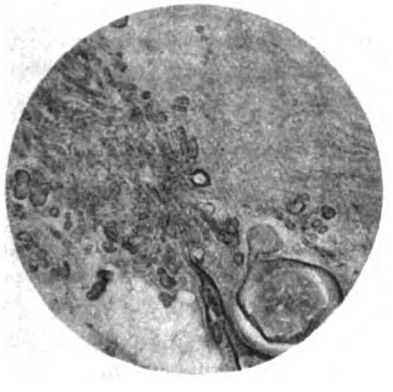


FIG. 2.—From an area of chronic mastitis in a lady forty-five years old, showing how the glandular apparatus is involved, as usual, in the inflammation, and how cysts are formed. In the largest cyst the epithelial lining is seen to be much and irregularly thickened.

The truth probably lies in quite another direction, that the female breast is especially liable to chronic inflammation during the period of involution. Hence the frequency of "involution" cysts during that period of life, and hence the greater liability to cancer then than earlier.

No cyst of the breast, even those which are not lined with glandular epithelium and are called lymphatic cysts, is a natural occurrence or a part of a natural occurrence. It is the result of disease, and the more clearly this is recognised and borne in mind the better for the patient.

Chronic mastitis may attack one breast, and may there form a single tumour or many indurations; or it may attack both breasts, either separately or at the same time. And it may attack the same breast at different times. There is not one of these conditions I have not seen examples of. Thus, I removed a single thick-walled cyst of the breast of a woman thirty-seven years old in 1886. In 1903 she was quite well, and had never had trouble either in that or in the other breast.

In 1887 I removed two cysts from the upper portion of the left breast of a lady æt. 35. Eight years later I removed one cyst from above the left nipple and two cysts from the upper and inner portion of the right breast. Seven years after the second operation she was quite well.

In October, 1893, I removed the axillary portion of both breasts of a lady æt. 46, on account of small cysts in indurated tissues, and she was well when I last heard of her in 1903.

In January, 1894—and this is an instance of peculiar liability to chronic inflammation of the breast,—I removed a single cyst from the lower part of the right breast of a lady between thirty-five-and forty years of age. In 1900 there was an indurated area at the extreme upper limit of the same breast, and when it was removed it was found to be composed of tiny cysts and indurated tissues. Fourteen months later I was obliged to remove a small collection of cysts in the substance of the

Digitized by Google

left breast. And quite recently I have been compelled to amputate the right breast for a more widespread recurrence of the cysts and induration there. Yet there was no appearance of cancer, either to the naked eye or to the microscope, in this last outbreak of the disease.

I believe the best hints in diagnosis, for I can give no more than hints, are that indurations due to chronic mastitis are much more often multiple than cancers are; that they are much more frequently associated with perceptible cysts; that they are much more likely to affect both breasts; that they are much more painful than cancer in an early stage. Indeed, the attention of the patient is, as a rule, drawn to chronic mastitis by the occurrence of pain in the breast; while cancer is, eight or nine times out of ten, discovered by accident, and is quite painless. I do not think that dimpling of the skin ever occurs in connection with chronic mastitis, but the glands in the axilla are frequently enlarged. The enlargement is often limited to a single palpable gland, and the gland is not fixed, and is at the most but firm, not hard. I need scarcely say there are none of the active signs of inflammation present in the breast, such as increased heat or redness of the skin.

The cases in which it is most difficult to distinguish between chronic mastitis and cancer are those in which the affection is limited to a single lump in one breast, where the firmness of chronic mastitis almost reaches hardness, and where there is one firm gland in the corresponding axilla.

Relation of chronic mastitis to cancer.—The relation of chronic mastitis to cancer has long been known to surgeons. It was pointed out by my great master, Sir James Paget, in the fourteenth volume of the 'St. Bartholomew's Hospital Reports' in the year 1878, more than twenty-five years ago (page 65). The microscopic characters of the disease had not then been the object of study, so that the masses were spoken of as "indurations." But Sir James Paget, with his customary intuition, divined them to be due to "some slow inflammatory or cirrhotic change." Since that time they have been studied largely in this country, and excellent accounts and illustrations of the microscopic characters may be found in the 'Path. Soc. Transactions' by Mr. Paul, Mr. Betham Robinson, Mr. Raymond Johnson, and others. The changes which mark them to be dangerous, and the transition from inflammation to cancer are shown in some of these illustrations.

I wish I could tell you in what way the transition in its earliest stages is apparent to the medical man. I wish I could describe the *precancerous* stages for the breast as I have been able to some degree to do for the tongue. It is doubtful whether it will ever be possible to do so in such a manner that they shall be perceptible either to the patient or to the medical attendant. The actual signs are no more than a little greater hardening, a little less mobility, and perhaps the smallest suspicion of dimpling of the skin, if the lump is not too deeply seated in the breast.

The following cases prove the difficulty of the diagnosis. In 1891 I was consulted by a maiden lady, nearly fifty years of age, on account of knotty induration of both breasts, with the hardest knot on the outer side of the right breast. Seven months later I saw her again, and was so ill-satisfied with the knot on the right side that I desired a consultation with Sir James Paget. He thought that all the knots were due to chronic mastitis. It was one of his old cases of induration, and he took the less serious view of this case because (as he had stated in his lecture), in his opinion, "If there be many, they are not likely to be or to become cancerous." But he quite agreed that it was sufficiently suspicious to call for removal. It proved to be typical carcinoma, and the glands were enlarged in the axilla; but I am

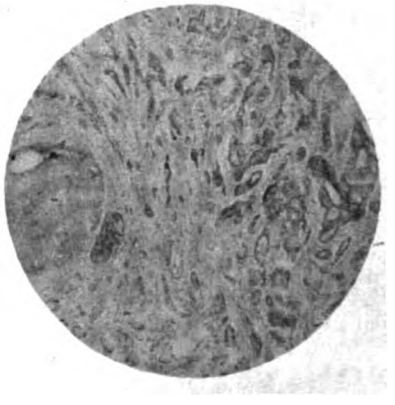


FIG. 3.—Showing the development of carcinoma in this case. On the left hand is a small cyst and, perhaps, a smaller cyst was forming close to it.

Digitized by Google

thankful to say that there has been no recurrence of the disease.

Several years ago I amputated the breast and contents of the axilla for typical cancer. Within a year of that time the daughter of the lady, about forty years of age and married, consulted me for an induration of the right breast above the nipple and for a smaller induration in the axillary portion of the left breast. I removed the affected portion of both breasts; they were both due to chronic mastitis; but in the middle of the harder and larger lump on the right side there was a small nodule of carcinoma. It was in the centre of the inflamed area, and its greater hardness was therefore obscured by the lesser hardness of the inflammation.

Chronic mastitis need not become cancerous.—It need not be imagined that every lump due to chronic mastitis must of necessity become in the course of time the seat of cancer. The disease may get well spontaneously, or the patient may recover from the attack under such simple treatment as support of the breast or, better still, of carefully adjusted strapping. I could give several examples in which this result has been achieved. Again, the disease may become quiescent, neither disappearing nor extending nor changing in any obvious fashion in the course of many years. I have recently had the opportunity of examining a portion of the breast in which this had occurred. In November, 1895, a young married lady, not thirty years of age, was brought to me with an induration of the outer part of the left breast, which had commenced a year previously, and had been attended with distressing pain. There was an enlarged firm gland in the axilla, but there was a gland equally large and firm in the other axilla, although the breast betrayed no sign of inflammation. I did not see her until the autumn of last year, eight years after the first consultation and nine years from the beginning of the attack. The conditions were precisely the same as they had been in 1895. But the outer portion of the affected breast was removed, and was found to consist almost entirely of fibrous tissue, in which the signs of inflammation could scarcely be discerned. This might well be regarded as a kind of cirrhosis, and was not likely ever to become the seat of carcinoma.

General rules for treatment.—I would that I could tell you which cases of chronic mastitis are likely to get well or to become "cirrhotic" and which are likely to become the seat of cancer in the course of months or years. But I cannot do so, for, indeed, I do not know. I can only impart to you the general rules of practice in all such cases which I have laid down for myself; and I believe that, in the present state of our knowledge, you cannot do better than follow them. They are very few and not difficult to remember.

Chronic mastitis, whether single or multiple, of one breast or of both, may be treated in young women by strapping. The strips of strapping should be long and broad. They should be carried from near the spine round the wall of the chest over the whole of the affected breast to above and below the other breast. If both breasts are affected they should each be separately strapped in the same manner. The strapping should be applied so as to produce firm and equal, but not painful pressure. And the breasts should be kept strapped for at least a month or six weeks.

FIG. 4.—Showing what may be termed the "cirrhotic" condition of the breast left in this case by chronic mastitis.

Chronic mastitis in older women which is of recent occurrence and obviously not malignant may be treated in the same fashion.

If there is a cyst large enough to be certainly diagnosed, it may also be strapped, but it will probably not be cured by strapping unless it has first been emptied of its contents by a fine trocar and cannula.

Tumours which are probably due to chronic mastitis, but which exhibit doubtful signs, especially if they are single and occur in women of more mature age, should be incised without delay, so that their real nature may at once be determined.

Digitized by Google

Tumours due to chronic mastitis which are not cured or very decidedly the better for treatment should not on any account be left indefinitely. A certain definite period should be assigned for treatment and, if the treatment has not proved successful at the end of that period, the disease should be removed without hesitation.

I generally allow six weeks, not longer; sometimes not more than a month. It is a common practice among consultants—and I was guilty of it myself until a few years ago, when I was rudely shaken out of it by one or two disagreeable surprises—to say to the patient or to the medical attendant that the case is not serious; that the induration is due to chronic mastitis, and that nothing need be done unless the tumour should grow larger or undergo some other change. In nearly every such case the responsibility of discerning the transitional changes is cast upon the patient or her medical attendant, a course which is most unfair; for the patient is quite incapable of judging of her own case, and the medical attendant, however skilful and careful he may be, rarely has seen a sufficient number of similar cases to be able to give an expert opinion in such difficult circumstances. Let me give an example. In October of a certain year quite a young married lady was referred to me by an insurance office. She would have been accepted as a first-class life had it not been for an induration in one breast, which had been there for a year or more. It had been examined in the spring by one of my surgical friends in town, of whose opinion I have such an esteem that I could scarcely imagine him to make a mistake. He had pronounced the tumour, or rather the induration, to be due to chronic mastitis, and had advised that it should be watched by the lady's medical attendant. This was regularly done, and there was a certificate from this gentleman to the effect that the disease was quite innocent, that it had been pronounced to be innocent only a few months previously by an expert, and that he was quite sure it had not altered in any respect during the months he had been watching it. Seeing the age of the patient it seemed to me very probable that this statement was correct, but I was quite shocked when I found a very much harder mass than is usual in chronic mastitis, and had to express the opinion that in all probability the lady was actually suffering from carcinoma. I begged her to go once more to the consultant who had seen her in the spring, and to ask him to remove the disease. She did so, and he was at once struck with the change which had taken place in the six months. When he operated he found, in the middle of a mass of chronic mastitis, a very small nodule which proved, to the naked eye and to the microscope, to be typical carcinoma. There can be no reasonable doubt that this nodule of carcinoma had developed during the few months which elapsed between the two consultations, but the development was so insidious that the changes which accompanied it were not evident either to the patient or her medical attendant.

Extent of operation.—Of the operation which is necessary I have no hesitation in saying that it suffices to remove the affected portion of the breast and, where both breasts are affected, to remove the affected parts of both, keeping well outside the disease. This has been my practice, and has been, I believe, the practice of most surgeons for many years. But lately there has grown up a new school which enjoins the removal of the entire breast in cases of chronic mastitis in which an operation is necessary. I have no sympathy with this advanced doctrine. I believe it to be bad surgery and worse policy. I understand it to rest on the pathological showing that all parts of such breasts are in an unstable condition, and that any part of any one of these breasts may become the seat of cancer. This may be so on theory, but it is not borne out in practice. I tested it during last year by following up my private cases during the last twenty years. With the very few exceptions in which amputation of the breast was performed for disease which involved every part of the breast, all the operations were limited to free removal of the affected parts, whether solid or cystic. There is not one of the patients who has since suffered from cancer of that breast. In several of them both breasts were affected simultaneously, or after an interval, short, or of many years' duration, so that it would have been more useful to the patient to amputate the sound breast at the first operation than to cut away the whole of the affected breast. When disease has returned a similar partial operation has been performed, save in one instance in which recurrence of the chronic mastitis was very rapid, very extensive, and very

Digitized by Google

threatening. In that case the breast was amputated and with absolute success, since the patient is now, at the end of seven years, perfectly well.

These sweeping operations for disease of limited extent which is not cancerous and which may never become cancerous are to be deprecated. Surgically they are not justified. Politically they prevent the very result we are most anxious to attain, for they deter women, especially of the higher social orders, from consulting us at the moment they find a tumour in the breast. The reward we hold out to them for doing so is a better result of operation if the disease is malignant, and no operation at all, or only a trivial operation if the disease is not malignant. But, if women are to be mutilated and disfigured in every case of tumour of the breast whether it is malignant or not, how shall we ever induce them to consult us as soon as they discover their trouble?

Serious Consequences of Deep Cervical Incisions in Labour.—Hofmeier describes two cases in which this method of facilitating delivery, first advocated by Dührssen, had very serious results. In the first case the incisions were made by another obstetrician three years previously. When the patient came under the author's treatment it was found that the cervical canal from its middle upward was the seat of a firm cicatricial stenosis. As no advance in dilatation was made after the patient had been having severe pains all night, a careful instrumental dilatation of the cervix was done, and the child perforated and extracted. In the other case the author himself, in 1897, incised the cervix, owing to its unyielding nature. The incision was very slight, but furious bleeding was set up which could be checked only after a rapid instrumental delivery had made the parts accessible. Two subsequent deliveries were uneventful, but the fourth was protracted, and owing to the rigid scar tissue the cervix and lower uterine zone were ruptured by the advancing head, and the resulting hæmorrhage cost the patient her life. If incisions must be made, the author recommends that they shall be antero-posterior and not placed laterally.—*Medical Record*, vol. lxv, No. 8.

FOUR LECTURES

ON

NORMAL AND ABNORMAL CONDITIONS OF THE BLOOD.*

By EDMUND CAUTLEY, M.D.Cantab., F.R.C.P.Lond.,

Physician to the Belgrave Hospital for Children and the Metropolitan Hospital.

Lecture I.

Gentlemen,—In a course of four lectures I propose to deal with normal and abnormal conditions of the blood, anæmia, with and without enlargement of the spleen, and affections of the thymus gland, occurring in infants and young children.

To obtain a sample of the blood, prick the lower surface or edge of the lobe of the ear with a triangular needle. The ear is very insensitive, and blood can be obtained in this way from a sleeping child. Wipe away the first few drops, and do not use for examination blood which has been obtained by squeezing the ear. The sample should be taken before food, to avoid error from the leucocytosis of digestion. From the blood make three preparations: (1) A blood-film in the usual way—by pressing a minute portion of blood between two cover-slips, separating the latter by a sliding movement, and drying them slowly over a spirit flame. (2) A fresh preparation in the following manner:—Take up some staining fluid, 0·2 per cent. methylene blue (Grübler) in 40 per cent. alcohol, in a Thoma-Zeiss pipette, and take a largish drop of blood on a glass slide. Mix gently by blowing the staining fluid on to the blood. The colour becomes a deep green. Put on a seven-eighth-inch cover-slip, and seal the edges with vaseline. In this mode of preparation the white corpuscles are undistorted by pressure. (3) Make a fresh preparation by taking a minute portion of blood on a cover-slip and dropping it gently on the slide, or by allowing it to flow by capillary attraction under a cover-slip fixed on three sides by wax to a glass slide. Examine this at once for blood-plates, rouleaux formation, and fibrin formation. A fair idea of the degree of anæmia, of deficiency of hæmoglobin, of poikilocytosis, and of leucocytosis will be obtained.

* Delivered at the Medical Graduates' College and Polyclinic.

Digitized by Google

Normal and Abnormal States of the Blood.

	Normal.				Secondary Anæmia.		Primary Anæmias.			Intermediate Anæmias.		
	Adult.	Fœtus.	New-born.	Modifications with age.	Mild.	Grave.	Pernicious anæmia.	Leukæmia. Lymphatic.	Leukæmia. Splenomyelogenous.	Anæmia splenica infantum.	Hodgkin's disease.	Anæmia splenica of Banti.
Specific gravity	1050 to 1059	—	1,062 to 1,066	Falls at first, normal at 2 years	Normal or a little lowered	May fall to 1,035	Down to 1,030	May fall to 28		Falls	Normal or low	Falls.
Percentage of hæmoglobin	—	Deficient	About equals that of mother	Deficient during first few months	Normal or a little lowered	May fall to 30	15—25	25—50		Down to 30	Normal or low	13—50.
Number of erythrocytes	4,500,000 to 5,000,000	—	5,000,000 to 6,000,000	Decrease during first year, then increase up to normal at eighth year	Normal or a little lowered	Decrease down to 2,000,000	800,000 to 2,000,000, or even down to 500,000	Falls to about half the normal		Below 3,000,000	Normal or low	May fall to 1,000,000
Varieties of erythrocytes	Typical red cells	All varieties, especially microcytes, "shadow corpuscles."		Abnormal forms disappear in a few days	Poikilocytes few or absent	Poikilocytes and occasionally myelocytes	Poikilocytes and megalocytes numerous, microcytes, "shadow corpuscles"	Variable degree of poikilocytosis		Poikilocytes, megalocytes, microcytes	Normal or shows the changes of secondary anæmia	Poikilocytes, myelocytes, microcytes.
Polychromatophilia	—	—	—	—	—	—	Marked	—	—	Usual.		
Hæmatoblasts	None	Normoblasts. megaloblasts		Disappear in a few days	Absent	Normoblasts, megaloblasts, karyokinesis	Many megaloblasts, normoblasts, karyokinesis	Rare	Very numerous	Megaloblasts, normoblasts, karyokinesis	Normoblasts in late stages	A few.
Number of leucocytes	10,000	—	Excess 18,000, "leucocytosis of the newborn"	Gradual decrease	Normal or increased up to 65,000		Normal or deficient; rarely in excess	Increased: a lymphocytosis	Increased: a myelocytosis	Up to 50,000	Normal	Normal or decreased.
Small lymphocytes	20—30	—	50—75	Gradual decrease.	If there is a leucocytosis the relative proportions are unaltered, or the polynuclear cells may be increased in undue proportion		A relative lymphocytosis in bad cases	Up to 97, almost all small	Decrease to 10—30	Usually normal.	May be increased in late stages.	No increase.
Large lymphocytes	4—8	—	6—14	Gradual decrease						Usually normal.		
Polynuclear cells	60—70	—	18—40	Increase				Down to 3	Increased up to 50	Usually normal, vary much in shape		
Eosinophiles	0·5—4·0	—	0·5—10·0	Decrease				Rare	Up to 5	Normal, deficient, increased		
"Mast-cells"	Normal up to 0·5 per cent.	—	—	—				—	Many in cases in adults	Present.		
Myelocytes	Absent	Absent	Absent	—	—	Occasionally present	Few	Few or absent	35—60	Often present	Occasionally present.	
Coagulation	—	—	Slow and incomplete	—	—	—	Feeble	Increased.				
Rouleaux formation	—	—	Deficient	—	—	Deficient	Absent.					
Colour	—	Darker during first few days of life.		—	—	Abnormally pale	Dirty pink to coffee colour	Like *café au lait*.				

Digitized by Google

Enumeration of corpuscles.—The red cells are counted in the usual way by means of a Thoma-Zeiss or Gower's hæmatocytometer. If it is decided to fix the red cells without distortion in order to look for poikilocytes, use a 1·0 per cent. solution of osmic acid, to which may be added 0·2 per cent. methylene green. The latter dye stains the nuclei of the white cells, and they are readily recognised. A most satisfactory method is to use as a diluting fluid Toison's solution (methyl-violet 0·025, sodium chloride 1·0, sodium sulphate 8·0, neutral glycerine 30·0, and distilled water 160·0). The solution should be filtered before use. Count five fields of thirty-six squares each, repeat the process with three different drops of the diluted fluid, and take the average of the two results which most approximate.

Remember to clean the pipette thoroughly with normal saline solution, water, alcohol, ether, and dry air. An aspirator attached to the tube will be found very useful for this purpose.

For counting white corpuscles use 0·33 per cent. solution of glacial acetic acid, Toison's fluid, or 0·2 per cent. methylene blue or methylene green in 40 per cent. alcohol.

Staining methods.—The most useful dyes for the differential counting of white cells are the Ehrlich-Biondi fluid (Ehrlich-Biondi powder 15 grs., absolute alcohol 1 c.c., distilled water 6 c.c.); Ehrlich's triple stain; Ehrlich's acid dahlia solution; Jenner's stain, a solution of a salt of methylene blue and eosin in methylic alcohol, which fixes and double stains; and eosin and methylene blue, either separately or mixed in the proportion known as Canon's dye. For differential counting to ascertain the percentages of the different varieties of leucocytes examine 1000 cells.

Estimation of hæmoglobin.—Use either Von Fleischl's or Gowers' hæmoglobinometer. These depend upon colorimetric methods, and are not very reliable. A fair estimate can be made by ascertaining the specific gravity.

Estimation of specific gravity.—Mix chloroform and benzol in proportions to yield a fluid of specific gravity of 1059, that of normal blood. Let a drop of blood fall into the mixture; if it sinks add more chloroform, if it rises to the surface add more benzol.

Finally, when the drop remains stationary, floating like a red bead in the mixture, the specific gravity of the mixture is the same as that of the blood. The lower the specific gravity the lower is the percentage of hæmoglobin, and *vice versâ*.

Nomenclature.—The following classification of the various corpuscular elements found in the blood is by no means a perfect one, but is useful for practical purposes. It is based partly on the origin of the different cells, partly on the properties and shape of the nucleus, and partly on the affinity of the cell contents for aniline dyes.

A.—Red Corpuscles.

1. *Non-nucleated.*—(a) *Erythrocytes*, or normal red corpuscles; (b) *macrocytes* or *megalocytes*, larger than normal; (c) *microcytes*, smaller than normal; (d) *poikilocytes*, irregular in shape.

2. *Nucleated, or hæmatoblasts.*—(a) *Normoblasts*, cells of the size and colour of an erythrocyte, but containing a deeply staining nucleus. They are usually regarded as immature erythrocytes. They are normally present in the marrow in small numbers, and after hæmorrhage in large numbers. (b) *Megaloblasts*, very large, 11—20 μ, nucleated red cells, circular, oval, or irregular in shape. The nucleus is very large, occupies most of the cell, stains feebly but evenly, and is usually surrounded by a white band, an empty space separating it from the encircling protoplasm. They are found in fœtal marrow and in the blood of grave forms of anæmia. (c) *Microblasts*, small nucleated red cells, consisting of a nucleus in a cell smaller than a normal erythrocyte. They are rarer than the other two varieties. (d) *Sub-varieties* and modifications of the above

B.—White Corpuscles.

1. *Basophiles, mononuclears, lymphocytes, or hyaline cells.*—They are usually regarded as the youngest form of leucocyte, and are further subdivided, according to their physical characteristics and supposed period of growth, into: (a) *Small lymphocytes*, 5—10 μ in diameter. These consist of a round nucleus about the size of an erythrocyte, surrounded by a narrow zone of faintly staining protoplasm. The smaller the nucleus is the more deeply it stains. These cells pass into the blood from the adenoid tissue, and, growing by increase in both nucleus and protoplasm, pass into the next variety. (b) *Large lymphocytes, or large hyaline cells*, 13—15 μ in diameter. They differ from the last variety in being larger and

Digitized by Google

paler. The nucleus is large, stains feebly, and is surrounded by a bigger zone of protoplasm. (c) *Transitional or intermediate forms.* These may have an indented or horse-shoe-shaped nucleus, may be oval in shape, and may present all intermediate forms as regards size. The lymphocytes are often called mononuclear because they rarely contain more than one nucleus. They are called hyaline cells because of the clearness of the zone of protoplasm; and basophiles because they stain with basic dyes. They are best distinguished from the subsequent varieties by the absence of granules from the faintly staining protoplasm.

2. *Neutrophiles, or finely granular oxyphiles, polymorphonuclear or polynuclear.*—The granules stain faintly with acid and neutral dyes, the latter dyes being really faintly acid. These are regarded as a full-grown type of leucocyte. They are large round cells, 9—10 μ in diameter, containing many fine granules and an irregular or multipartite nucleus. The nucleus is twisted, variegated in shape, and often causes the cell to appear multinucleated. Hence the name polynuclear, though strictly it should be polymorphonuclear. The nucleus is situated in the centre of the cell, not being in contact with the cell wall, and stains deeply and unevenly with basic dyes. The granules are much smaller than eosinophilic granules, and surround the nucleus. The protoplasm does not stain with methylene blue.

3. *Eosinophiles, or coarsely granular oxyphiles.*—These are cells containing large granules which stain with Ehrlich-Biondi fluid and with acid dyes, such as eosin and acid fuchsin. They are regarded as the old or over-ripe leucocytes. They are usually rather smaller and more irregular in shape than the polymorphonuclear cells, and contain a similar nucleus, which stains rather more evenly and less deeply. The granules are large, uniform in size, and only stain well with acid dyes. Sometimes only the granules can be seen. The protoplasm stains with methylene blue. They are the most actively amœboid form of leucocyte. They are numerous in the thymus and marrow, and undergo division in the latter.

4. *Basophilic "mast-cells."*—These cells resemble the eosinophiles, but the granules do not stain with Ehrlich-Biondi fluid, and appear as clear white spots. The protoplasm stains mauve or purple with methylene blue, but does not stain with methylene green. They occur in very small numbers—0·25 to 0·5 per cent. or not at all—in blood, and are not uncommon in leukæmia.

5. *Myelocytes, or marrow cells,* can only be recognised by Ehrlich-Biondi's staining. They are spherical cells, differing from the large lymphocytes in containing a large number of granules. The granules are neutrophilic and resemble those found in the polynuclear cells, but the nucleus differs in being large, spherical or egg-shaped, and eccentric, almost filling and in close contact with the wall of the cell. It stains evenly and moderately. The cells are usually larger than the large lymphocytes though some may be smaller. They have no amœboid movement. They are sometimes supposed to be intermediate forms between the large lymphocytes and the polynuclear cells. Normally they are found in the marrow, but their presence in the blood is abnormal. Many in the marrow are eosinophilic; so too are a few of those which are found in the blood. They readily appear in the blood of infants.

Blood in the fœtus and the new-born.—The total amount of blood in the new-born child is proportionately less than in the adult, unless the so-called reserve blood is pressed out of the placenta, when it is greater. The reaction is alkaline, and the colour is rather darker during the first few days of life. The composition depends largely on that of the maternal blood, and is not influenced by age or sex. The specific gravity is greater than that of adults—1062 to 1066. It gradually falls during the first few weeks. From the age of ten weeks up to two years it ranges from 1048 to 1052. Hock and Schlesinger state that it reaches the normal average by the end of the first year. It depends directly upon the amount of hæmoglobin present. The hæmoglobin varies in amount at different stages of development. It is deficient in the fœtus, but at the end of pregnancy about equals that of the maternal blood. It usually decreases during the first few weeks of life, remains stationary up till about the sixth month, and then slowly rises. The percentage is generally lower in young children than in adults, being higher in boys than in girls. The hæmoglobin is readily disintegrated from the red cells, and passes into the plasma, leaving numerous "shadow corpuscles" in the blood. These latter are delicate pale rings, the stroma of cells from which the hæmoglobin

Digitized by Google

has passed. They were first described by Ponfick. Oxygen appears to be more firmly combined with hæmoglobin than in the adult. Milne Murray found that it required seven times as much reducing agent to get a reduced spectrum. The effect of the disintegration of hæmoglobin is to produce hæmoglobinæmia, and, if diseased processes are set up, it may produce hæmoglobinuria. Under normal conditions the hæmoglobin is converted into biliary pigments in the liver.

Coagulation is incomplete. Although the blood coagulates at the moment of birth, the clot forms slowly. This probably depends upon a deficiency in fibrinogen, which is stated by Scherenziss to be only about one third of that found in adults. According to Silbermann, the fibrin ferment is in excess, and this excess predisposes to disease. Salts are in excess compared with the amount in the maternal blood. There is a marked preponderance of chlorides in the serum, and there are more sodium and less potassium salts than in the adult.

Erythrocytes.—Just as the amount of blood is increased in the infant by delaying ligature of the cord until all pulsation has ceased, so too is the proportion of red corpuscles increased. There is an excess over the number in the maternal blood varying from 350,000 to 1,000,000 per cubic millimetre. During the first few days after birth, while the weight is decreasing, the number remains stationary or may even increase. After this it slowly diminishes during the first year, and attains to normal about the eighth year. The size of the cells varies within wide limits, and microcytes are more numerous. The cells are more spherical in shape, and have not the same tendency to rouleaux formation. "Shadow corpuscles" are often seen, and are more numerous if the infant's general condition is disturbed during the first few days of life.

Hæmatoblasts.—Normoblasts and megaloblasts are present in fœtal blood until the later months of pregnancy. They are often found on the first day of life in considerable numbers, and disappear by the end of the fourth day. They are most often found in premature infants. Anæmia and other pathological conditions may lead to their presence in the blood during the first few months of life much more readily than in older children.

Leucocytes.—An increased proportion of white cells is always present at birth, and is spoken of as the "leucocytosis of the new-born." Roughly the number is about double that found in adults. The same varieties are present as in adults, but the lymphocytes or young cells are much more numerous, and the polynuclear or adult cells are much less numerous. During the early days of life the percentage varies greatly, and the "leucocytosis of digestion" is much more marked. Not until the end of the first year does the number come down to that found in the adult. According to some writers the number does not reach the normal until the fifth year. At birth the cells are viscid, deliquescent, easily destroyed, and tend to adhere and form rouleaux. Eosinophiles may be deficient or in excess.

Differential Count of Leucocytes, Normal Blood.

	Adult.	Infant.
Small lymphocytes . .	20—30	50—75
Large lymphocytes . .	4—8	6—14
Polymorphonuclear cells .	60—75	20—40
Eosinophiles	0·5—4	0·5—10

The main differences are the greater proportion of young leucocytes or lymphocytes and the smaller proportion of old adult or polynuclear cells in the infant. In the third year of life the lymphocytes and the polynuclear cells are about equal in number, and about the eighth year the percentage composition resembles that of the adult.

Abnormal states of the blood.—The state of the blood varies as the development of the child. Hence all those conditions which retard the proper development of the child have a similar effect on the development of the blood. Consequently under such conditions the blood is normally like that found in younger infants. Thus a mere examination of the blood is not sufficient, in order to ascertain the presence of maldevelopment or disease, unless it is found that the characteristics of the blood do not conform with those normal for the age of the child. A leucocytosis is normal in infancy, but indicates disease in later life. A lymphocytosis is normal in infancy, but after three years of age becomes abnormal.

Digitized by Google

The blood is much more sensitive to morbid influences than in adults, marked leucocytosis and grave anæmia being produced by much slighter causes. The most common blood-change is that due to anæmia, and practically under the heading of anæmia may be included all abnormal states of the blood. It is one of the commonest symptoms of infantile disease, and is produced more readily in infants than in adults. Thus we find it due to unsuitable food, impure air, and bad hygienic surroundings. It may follow gastro-enteric affections, rickets, tuberculosis, or inherited syphilis. Most of these affections cause a reversion of the blood to a more infantile type, such as lymphocytosis and the reappearance of deformed erythrocytes and hæmatoblasts. Hence the characteristics of the blood must be regarded as dependent upon the anæmia and delayed development rather than as a special feature of the particular disease.

The specific gravity is lowered in all forms of anæmia, and may sink very low, down to 1028, in pernicious anæmia and leukæmia and in tuberculous meningitis. It usually falls in nephritis. It may be raised in cardiac affections. The percentage of hæmoglobin may vary from day to day. It may sink to 15 to 20 per cent. in pernicious anæmia and leukæmia, and it also falls considerably in severe cases of intermediate and secondary anæmia.

The erythrocytes diminish in number in most acute and chronic diseases, especially in those producing anæmia. Poikilocytosis and polychromatophilia are seen in bad cases of primary and, occasionally, secondary anæmia. Hæmatoblasts readily reappear, even in the secondary anæmias of young infants. According to Cabot, the changes in the blood in secondary anæmia occur in the following order:—(1) Deficiency of hæmoglobin, fall in specific gravity; (2) poikilocytosis, reappearance of macrocytes and microcytes, polychromatophilia of red cells, loss of rouleaux formation, appearance of shadow corpuscles; (3) actual deficiency in red-cells; (4) normoblasts and megaloblasts, possibly indicating regenerative changes.

It is important to understand clearly the meaning of the following terms:

1. *Leucocytosis.*—By this is meant an increase in the number of leucocytes in the blood without any relative decrease in the proportion of the polynuclear cells. The percentage of the latter may be increased. It is more exaggerated in infants than in adults, no matter what the cause may be.

2. *Lymphocytosis.*—By this we mean that there is a relative increase in the number of lymphocytes or young leucocytes, with or without an actual increase in the total number of white cells. It is the great characteristic of infants' blood. It occurs normally in all healthy infants and in all disorders which retard its general development. It is present in rickets, inherited syphilis, scurvy, and various forms of anæmia in which there may be no leucocytosis.

3. *Eosinophilia* means that there is a relative increase in the number of the eosinophile cells. It may be regarded as normal in infants, for the proportion may reach 10 per cent. of the total. It is occasionally found in leukæmia, bone disease, gonorrhœa, and at puberty in girls. It is common to certain skin diseases, such as pemphigus, dermatitis herpetiformis, and urticaria.

The presence of leucocytosis is sometimes useful for diagnostic purposes, as shown in the following table:

Physiological.	Absent.	Present.
In the new-born.	In early phthisis.	Advanced phthisis.
During digestion.	Miliary tuberculosis.	Secondary infection.
After cold baths.	Meningitis.	Septic meningitis.
After massage.	Caries of bone.	Cerebro-spinal meningitis.
After exercise.	Joint disease.	After hæmorrhage.
In the dying (not constant).	Pleurisy.	Pleurisy.
	Pericarditis.	Pericarditis.
	Peritonitis.	Septic peritonitis.
	Enteric fever.	Gastro-enteritis.
	Measles.	Scarlet fever.
	Rötheln.	Variola.
	Mumps.	Diphtheria.
	Influenza (generally).	Tonsillitis.
	Malaria.	Bronchitis.
		Pneumonia.
		Rheumatic fever.
		Pyæmic conditions.
		Septicæmic conditions.
		Osteo-myelitis.
		Erysipelas.
		Local abscesses.
		Appendicitis.
		Rickets.
		Skin diseases.
		Some affections of liver and kidneys.
		Leukæmia and allied affections.

The above table shows that leucocytosis is

Digitized by Google

present in most febrile disorders and in most septic processes. The polynuclear cells may be increased up to 90 per cent. of the whole number of leucocytes. In febrile conditions it is noteworthy that leucocytosis may appear before the fever, and bears no definite relationship to its severity, nor does it always occur with it. In pneumonia a "blood-crisis" may precede the temperature crisis.

The value of a blood examination for diagnostic purposes is shown in the table. Thus it may enable a differential diagnosis to be made between scarlet fever and measles, or between enteric fever and gastro-enteritis. It is important to remember the "leucocytosis of digestion," which is at its height in about two hours after a meal, or an erroneous conclusion may be drawn. Leucocytosis may also result from drugs, such as antipyrin, pilocarpin, quinine in poisonous doses, salicylates, and others, and may follow the injection of tuberculin or thyroid extract.

Leucopenia, or diminution in the number of white cells, may be found in some of the infective diseases in which leucocytosis is absent, such as enteric fever, measles, influenza, and uncomplicated forms of tuberculous disease. In the later weeks of enteric fever it is almost invariable, and is associated with lymphocytosis. The number of white cells may also be diminished by starvation or malnutrition.

Myelocytes.—The presence of myelocytes in the blood must always be regarded as abnormal. They are most frequently found in pernicious anæmia and in spleno-myelogenous forms of leukæmia. They have at times been found in other morbid states, such as diphtheria, caries of bone, septicæmic conditions, Addison's disease, Hodgkin's disease, rickets, and anæmia splenica infantum.

The Assimilation of Sugar during Childhood. — Greenfield ('Jahrb. f. Kinderhk.,' vol. lviii, No. 4) studied this subject in thirty-three children, aged from one month to thirteen years. No case which ran a fever temperature was studied, but convalescents were taken. The results showed that the limits of assimilation of grape sugar are not influenced by the body weight, by the state of nutrition, nor by such diseases as anæmia, rachitis, tuberculosis and lues, and enlargement of the liver and spleen. It is dependent, on the contrary, upon the age, increasing with each year, until at the end of the first decade it reaches the amount which is normal for adults.—*American Journ. of Obstetrics.*

WITH DR. CHARLES W. CHAPMAN'S POST-GRADUATE CLASS

AT THE

NATIONAL HOSPITAL FOR DISEASES OF THE HEART, SOHO SQUARE.

GENTLEMEN,—Here is a young woman æt. 21, whose home is at Harringay, and who works in the city as a book folder. She is due at her work at 8.30 in the morning, consequently she should get up very early so as to have time for a good breakfast, but as a fact she has been in the habit of lying in bed up to the last moment, then, after swallowing some tea, hurrying to the station (which is a quarter of an hour's walk up hill), with a piece of bread and butter in her mouth. On arriving at King's Cross there was a twenty minutes' walk, at the end of which she had to get up three flights of stairs as best she could. All this exertion without having taken food since the previous evening. Furthermore, the middle day dinners generally consisted of a little fish and bread, or at times only pastry. This is a type of the life of thousands of young women working in London.

Her father is living, but he has always been delicate; her mother died of heart disease. The personal history does not help us, for though never robust the patient has never been laid up.

She complained of attacks of faintness, palpitation, dyspnœa on exertion, vertigo, cough, and cardiac pain. Six months since while carrying a heavy parcel across the work-room she had an attack of hæmoptysis.

On inspection you will notice there is no special fulness of the cardiac region. The dulness is normal on the right side, upwards to just above the second rib, and to the left one and a half inches outside the nipple line. The apex is in the fourth space in the nipple line. There is a thrill presystolic in time over the apical region. There is no marked epigastric pulsation. I am purposely deferring auscultation until the last because this part of the investigation should in many cases be of the nature of what chemists call a confirmatory test. In chemical analysis the presence of numbers of substances is negatived by a graduated process, till by a process of exclusion only one or two substances are left. It is at this point that by means of confirmatory tests a definite conclusion is arrived

Digitized by Google

at. On these lines in the case before us we inferred that the lesion was not of very long standing or of a severe type, because of the absence of undue prominence of the cardiac region; and, to a certain extent, by the general appearance of the patient. The shape of the dulness was suggestive of mitral rather than aortic disease, and the thrill at the apex was a sign pointing to the probability of stenosis of the mitral valve. Auscultation gave the confirmatory test.

As regards drugs:—From the conclusions arrived at from a study of the case in all its bearings, it was considered that rest, careful feeding, and tonics were best calculated to improve the general health, and with it the cardiac muscle, and that strychnine was more suitable than digitalis. [Under this *régime* the patient has made uninterrupted progress.]

It may be asked why a patient with so slight a lesion was admitted? She had been for some time under treatment in the out-patient room with a steady increase in severity of the symptoms, and it was evident she would only go from bad to worse unless she had rest in addition to medicinal treatment. The points always to be aimed at in our endeavours after success in treatment are accuracy in diagnosis and an anticipation of possible complications.

This next case is somewhat similar to the one we have just seen, but the history is more definitely rheumatic. She had scarlet fever when a child, frequent sore throats (tonsillitis?), flying pains "all over her," and influenza three times. She has had palpitation and occasional faintness for two years, and her ankles were at one time swollen. She has always been anæmic and her menstruation deficient in amount and quality. The cardiac dulness extended from the right edge of the sternum to the left anterior axillary line and upwards to the third rib. There was a pre-systolic thrill and a rumbling murmur during the same period. The second pulmonic sound was not accentuated, but was reduplicated. The liver was down to the umbilicus. The lungs were unaffected, and the urine was normal.

Here we have four ætiological factors to take into account—scarlet fever followed by frequent sore throats, rheumatic pains, persistent anæmia, and repeated attacks of influenza.

(This patient died in the course of a few days. At the post-mortem the right auricle and ventricle were found hypertrophied and dilated; there was some obstruction at the tricuspid valve, while the mitral orifice was contracted to a small button-hole. It was clear that the enfeebled state of the right heart accounted for the absence of accentuation of the pulmonic second sound.)

The next patient, æt. 21, is by occupation a packer. Both parents are alive, and one brother has heart disease. She had rheumatic fever at thirteen years of age, which kept her in bed four months. Three months after convalescence chorea came on. She has always been pale. The ankles swelled two years since. The chief symptoms on admission were palpitation, orthopnœa, cardiac pain, cough, and loss of appetite. The cardiac impulse was feeble, the dulness extended to over an inch to the left of the nipple line, and the apex-beat was at the upper part of the sixth space three-quarters of an inch outside the same line. The action was heaving. You will notice there are double murmurs at both aortic and mitral areas, also that the aortic diastolic has its region of maximum intensity at the left sternal margin of the third and fourth spaces. The difference in the rhythm of the two sets of murmurs is very evident within a comparatively small area, for you will notice on moving the stethoscope a little lower and further to the left the pre-systolic mitral takes the place of the diastolic aortic, while the mitral systolic appears to occupy that of the aortic diastolic. The liver is down nearly to the umbilicus, and there are crepitations at the bases of both lungs, with impaired resonance of the right lung posteriorly—indications of long-standing disease of the heart with failure.

This little girl, æt. 10 years, illustrates not only the existence of serious cardiac lesions in a young child, but also the comparative efficiency of compensation possible in young people. She belongs to a large family, several of whom have heart disease (a brother is in the hospital at the present time), and she herself had rheumatic fever at six after pneumonia, since which she has frequently suffered from rheumatic pains. She complained of cardiac pain, palpitation, and dyspnœa, all increased on exertion. On inspection we notice that the patient is pale, that the carotids pulsate visibly, and though the cardiac impulse is not remarkably strong, yet it is more pronounced than

Digitized by Google

in a healthy heart. The cardiac dulness is increased in all directions, and the apex-beat is in the fifth-space nipple-line. You will notice by these tracings that the one taken a fortnight after admission shows considerable recession of the cardiac dulness, especially on the left side, which, instead of extending to the mid-axillary line, reached only the anterior one. The murmurs are systolic and diastolic aortic, and both presystolic and systolic mitral. You will notice that both aortic sounds are audible, showing—as regards the second sound—that the sigmoid valve is acting, though but imperfectly. The presystolic is probably a "Flint's" murmur, and not due to actual mitral stenosis. She is doing very well, and I daresay will continue to do so if she is fortunate enough not to have any further rheumatic attacks.

Here is a man æt. 37, but having a much older appearance. He is described as a labourer, though he was until recently in the army. On admission it was noted that his nutrition was fair. He complained of severe pains across the chest and down the left arm, dyspnœa on the least exertion or excitement, cough with expectoration occasionally streaked with blood, and swelling of the legs. His expression had the anxious look so common in cases of aortic regurgitation accompanied by anginal symptoms. There was visible pulsation of the carotids, especially of the left. Pulse 90, regular, and of low tension. The family history was of no significance. He had malaria in India at 19 years, syphilis at 27, rheumatic fever at 29 (six weeks in bed), malarial fever in South Africa, during which illness he first developed cardiac symptoms. It was at this time that valvular disease was discovered, which caused him to be invalided home. Before being sent to the front he was examined and pronounced medically fit for active service. It is presumable that the cardiac signs and symptoms were at any rate not well marked at that time. He does not allow that he drank excessively in the service.

On examination the heart was found to be much hypertrophied and dilated, the apex beat in the sixth space and considerably to the left of the nipple line; the latter, moreover, had but little "push." The liver was not much enlarged. There were a few moist sounds at the bases of the lungs. Urine 1030, devoid of albumen and sugar.

First as to the actual state of the heart, then of the cause or causes of the lesions and the recent breakdown. The physical signs can in this case be very definitely interpreted. They indicate incompetence of both aortic and mitral valves with some obstruction at the former. It is, however, the condition of the aortic valve to which I would enlist your particular attention. The diastolic murmur is a very long one, and exceeds the systolic in the proportion of nearly three to one. From this we infer that the valve has practically lost its function. It may be dangerous to generalise when discussing the interpretations of physical signs, but as a broad rule one may say the gravity of a case of aortic disease is proportional to the lengths of the two murmurs. If the systolic is harsh and full while the diastolic murmur is little more than a whiff, we may conclude the obstruction is of a kind that a strong left ventricle will be fairly equal to, and that the regurgitation is, for the present at any rate, not of very serious import. If, on the other hand, we meet a state of things such as exist in this patient, we infer that the aortic valve is practically useless, and our prognosis is accordingly bad. This is especially the case when the second sound is completely replaced by the murmur.

The causes of the cardio-vascular disease you see here are no less than four, if not five—malaria, syphilis, rheumatic fever, recurrent malarial fever, and probably alcohol. It is significant that until he had the fever in Africa he had had no cardiac symptoms; in fact he had no fault to find with his health. Probably whatever damage the rheumatic attack did, a state of fair compensation had been not only established, but maintained. The hardships of campaigning in Africa, such as had to be endured during the late war, coupled with a second attack of malarial fever, doubtless told upon the nutrition of the myocardium, leading on to further dilatation of the cavities, syphilis and alcohol helping the heart on the downward grade.

The man has been treated by entire rest, increasing doses of iodide of sodium, arsenic and strychnine. The angina gradually disappeared, the cardiac impulse has become stronger, and the general nutrition much improved. He is a permanent cardiac cripple, and endeavours are being made to obtain employment suitable to his state.

This patient was in the hospital from May to July this year, and I asked him to come to-day to

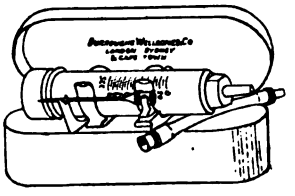
Digitized by Google

show you how some of these desperate cases improve under rest and treatment.

J. L—, æt. 18, had rheumatic fever at 11 years, from which he recovered without having any special symptom. A second and less severe attack at 15, a third one two and a half years afterwards, and again six months ago. He had but little trouble with his heart until the last attack, after which he had dyspnœa following severe exertion, giddiness on walking, and latterly excruciating pains across the chest. The man's expression was a very anxious one, and he was pale and thin. There was marked prominence of the cardiac region, the apex-beat was diffused in the fifth and sixth spaces, one and three-quarters inch to the left of the nipple-line; the cardiac dulness extended more than two inches in the same direction, upwards to the third rib, and to the right one inch beyond the sternal margin in the fourth space. There were well-marked aortic, systolic, and diastolic murmurs, and a systolic mitral. The second aortic sound was present, but it was very faint. The case was then described as "double aortic, mitral systolic heart-failure with marked dilatation, and accompanied by anginal attacks." On admission a case could scarcely look more unpromising. He could not lie down and, after dosing, he would wake up in a fright from urgent dyspnœa, yet he gradually improved, and the apex-beat became more definite. His heart is still enormous, but it is vigorous. You will remember the second sound was very faint when he was admitted, and you can hear that is still the case. It is clear that the incompetence of the aortic valve is pretty nearly complete, and his improvement is entirely due to increased strength of the heart muscle, especially of the left ventricle, which is now able to drive on enough blood so that, after the reflux has done its worst, a fair amount of it is sent into the systemic circulation. This is shown by the improved state of his nutrition and the absence of giddiness and faintness. The throbbing of the rapidly filled and collapsing vessels causes unpleasant beating, especially in the head; but this he must put up with, for it is his salvation. Of course the prognosis in such a case is always grave, for a sudden and fatal failure may occur at any moment. Some little extra strain, a flatulent stomach, or emotional excitement is sufficient to cause fatal syncope. On the other hand, the most unpromising cases do rally over and over again if judiciously managed. There is no symptom too trivial for the medical attendant's consideration if he is to make the best of the small margin of cardiac power present in these cases.

Weymouth Street, W.,
March 14th, 1904.

The B. W. and Co. All-Glass Aseptic Serum Syringe.—Serum treatment having become an established principle of modern therapeutics, a serum syringe is now an essential item of a complete medical equipment. The importance of a scrupulous avoidance of septic contamination of serum is obvious, and the employment of a simply constructed syringe which can be readily sterilised is, therefore, highly desirable. The necessary requirements are fulfilled in the B. W. and Co. All-Glass Aseptic Serum Syringe, which is constructed on principles precisely similar to those of the B. W. and Co. All-Glass Aseptic Hypodermic Syringe. The syringe itself is made entirely of



glass, and consists of three simple parts—barrel, piston, and nozzle, which are instantly detachable and easily sterilised. The adoption of a solid piston which fits the barrel with absolute precision renders unnecessary the use of leather or other packing. The needle is attached to the nozzle by a flexible rubber joint, which guards against injury from sudden movement on the part of the patient. The B. W. and Co. All-Glass Aseptic Serum Syringe is manufactured in five sizes—of 2 c.c., 3 c.c., 5 c.c., 10 c.c., and 20 c.c. capacity respectively. Each syringe is issued in a metal case, and is accompanied by two steel needles. If platino-iridium needles are desired they can be fitted to order.

Sublimate Injected into the Kidney.—The direct injection of a solution of sublimate into the parenchyma of the kidney of rabbits causes a deposit of calcareous salts around the spot.—*Journ. A. M. A.*, vol. xlii, No. 8.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 595. WEDNESDAY, MARCH 23, 1904. Vol. XXIII. No. 23.

CONTENTS.

PAGE

*A Clinical Lecture on some Common Symptoms of Disease in Children and their Diagnostic Significance. By ROBERT HUTCHISON, M.D., F.R.C.P. 353

*Four Lectures on Normal and Abnormal Conditions of the Blood. By EDMUND CAUTLEY, M.D., F.R.C.P. LECTURE II.—Primary and Secondary Anæmias 362

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. J. H. Booty & Son, 30, Holborn, E.C. Telephone 1717, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London E.C.*

A CLINICAL LECTURE

ON

SOME COMMON SYMPTOMS OF DISEASE IN CHILDREN AND THEIR DIAGNOSTIC SIGNIFICANCE.

Delivered at the Hospital for Sick Children, Great Ormond Street, W.C.,

By ROBERT HUTCHISON, M.D., F.R.C.P.,

Assistant Physician to the Hospital and to the London Hospital.

LADIES AND GENTLEMEN,—Those of you who have attended the out-patient department here must have been struck by the fact that the number of actual complaints for which children are brought to the hospital—that is to say, the things noticed wrong by the mother—are comparatively few in number. And that, after all, is inevitable. A young child is unable to express—even supposing he is able to experience—those multifarious abnormal sensations which make up what we call the symptoms of disease in grown-up people. And it follows from this that your diagnosis must always rest mainly upon the results of your objective examination. Now, I need hardly tell you that therein lies one of the great difficulties of children's practice—you get so little help from your patient. The more, therefore, is it necessary for you to study as carefully as you can such symptoms, few though they be, as the friends of the child have been able to observe. I thought therefore that we might profitably spend our time this afternoon if we took up together the study of the commoner symptoms which children present, and tried to arrive at some idea of their value in diagnosis.

Wasting.

The first symptom that I wish to direct your attention to is one which occurs with very great frequency in the out-patient department. It is

Digitized by Google

that spoken of as *wasting*. With regard to this symptom, I would say first of all that it may be due on the one hand to very serious organic disease or, on the other hand, to comparatively trivial disorders of health. And what you have always to do when a child comes before you presenting wasting as one of its chief symptoms, is to eliminate so far as you can the possibility of organic disease before you go further. In the case of young infants there are three organic diseases which are specially apt to be accompanied by wasting as their chief symptom. The first of these is *congenital syphilis*, the second is *tuberculosis*, and the third is a *latent empyema*. With regard to the first of those, there should be no difficulty; you should easily be able, by inquiring into the family history, and by examining for such symptoms as snuffles, craniotabes, rashes, and other manifestations of inherited specific disease, to recognise that it is syphilitic or exclude that disease as a cause of the wasting. If you are still in doubt, you will often find that by the therapeutic test—that is to say, by the administration of grey powder—you can tell within a very short time whether such a taint is present or not; for syphilis may cause wasting even though present in only a small degree. With regard to *tuberculosis* in young infants it is very much more difficult to be certain; in fact, I would say that often you cannot be certain of it at all, because tuberculosis may have attained a very widespread degree of development before it produces any pronounced physical signs. Often in such cases you must remain in doubt, and watch the effect of treatment. If, under treatment directed to the symptoms, the wasting still continues and becomes progressive, then your suspicion that you are dealing with a case of tuberculosis is of course greatly strengthened. But as a matter of fact it will sometimes be only on the post-mortem table that the nature of the case will be made clear. There is nothing more difficult than to diagnose accurately between a case of tuberculosis in a young infant and one of so-called simple marasmus. With regard to the third of the conditions I mentioned, *latent empyema*, you should have no difficulty. You cannot miss it if you will examine the child carefully and systematically, particularly of course at the bases of the lungs. Not long ago a child was sent here with the history that he had suffered from measles a month or two previously, and that he had been wasting ever since. He had been under treatment outside, but had made no progress towards recovery, but went from bad to worse. He was sent here under the impression that he was suffering from tuberculosis, and it was thought that nothing further could be done. On examination one found marked dulness at one base, with signs of fluid. On putting in a needle pus was obtained, and after its evacuation the child at once improved, and made an uninterrupted recovery. So there is the chance that even where a child is under skilled observation an empyema may be overlooked, because it may have produced no sign other than that of continual and progressive wasting.

Of the non-organic diseases which may produce wasting as one of their chief signs in young infants, the principal are disorders of digestion and unsuitable feeding. As a matter of fact, these are by far the commonest causes of this symptom; either the child does not get enough nourishment, or he gets it in an unsuitable form, with the natural consequence that in both cases continuous loss of weight is manifested. Hence in the majority of such cases your chief duty will be to inquire into the nature of the feeding and of the food given, and to rectify any errors which you find.

When you come to inquire into the causes of wasting in older children you will find again that there is one great organic disease which is particularly apt to produce that symptom, and I need hardly say I refer to tuberculosis. Some of you saw among my out-patients this morning a child, æt. 3½, who was brought with a history of wasting; nothing else had been noticed except that the child sweated at night. On examination, however, we were able to find without difficulty unequivocal signs of fairly widespread tuberculosis. The commonest functional disease which you are apt to mistake for tuberculosis is that chronic form of dyspepsia which occurs in children at about the period of second dentition, and to which I am so often drawing your attention in the out-patient room. You know the history—the child is sent up with the statement that he has wasted, that he has a cough, that he has no appetite, and perhaps that he has certain nervous disturbances, such as headache or restlessness at night. Such cases are constantly diagnosed as tuberculosis when they are not so at all. And the mistake, after all, is not an unnatural one, for

Digitized by Google

there are many signs connected with them which in the adult one is accustomed to associate with phthisis; the child has been losing weight, has lost appetite, and has a cough. Those who see little of children and much of adults are apt to conclude that these signs mean phthisis in a child, just as they would rightly make one suspicious of phthisis in a grown-up person. You should know that many such cases are simply examples of chronic gastric catarrh associated with pharyngitis or enlargement of tonsils or adenoid growths, and they can be perfectly cured if they are put on a suitable diet and the condition of the throat attended to.

Cough.

I will next pass to the consideration of another symptom which is particularly common in children, especially at this time of the year, and that is *cough*. Of cough as a symptom in children I would say first that it would be a mistake to suppose that it is always, or indeed usually, the result of respiratory disease. The majority of coughs in children are not due to any abnormal condition of the lungs or bronchi at all. It is all the more necessary that you should be warned of this, because frequently the mother makes the diagnosis herself, and instead of saying the child is suffering from cough she will say he has "bronchitis," and so you are apt to be misled. Statistics were collected by Dr. Parkinson* from one of the children's hospitals in London on this subject, and he found that of a large number of consecutive cases of cough, fully 40 per cent. were due not to disease of the respiratory organs, but to abnormal conditions in the throat, whereas only 31 per cent. were due to disease in the chest. So you see there was actually a clear majority of cases in which the cough was due to the throat. I think there is no more important point to realise about cough in childhood than that. The *abnormal conditions of the throat* which may give rise to cough in children are three in number. You may have simple pharyngitis, or enlargement of the tonsils, usually existing with adenoids, or elongation of the uvula. These three conditions may occur together, as I need hardly say, or you may have combinations of any two or all three of them in any one case. Now, from the character of the cough you can often infer what is likely to be its cause in any particular case which comes before you. For instance, in *simple pharyngitis* it is usually a dry tickling cough, occurring at frequent intervals. In *adenoids* it is rather of a choking character, often accompanied by vomiting, and frequently also by epistaxis. On the other hand, the cough which is associated with an enlarged uvula is characterised by the fact that it comes on when the child lies down, because the uvula falls back into the pharynx and irritates the posterior wall.

When you come to consider cough arising from irritation in the bronchi you will often have to face the problem, Is it simple bronchitis or is it whooping-cough? Now that is a question which it is not always easy to answer, although it is one to which the mother expects you to be able to give an exact and definite reply. Although you cannot be certain always about the existence of *whooping-cough*, there are certain things which should make you suspicious of it. And first, the cough of whooping-cough is essentially paroxysmal, it tends to occur especially at night, and it is often followed by vomiting. When you examine the child you find that the cough is out of all proportion to the severity of the physical signs. There may be only a few bronchitic signs in the lungs, although you are told that the cough is one of very great severity. You will frequently find also that there is a sublingual ulcer, which is of real value because it may occur before the cough has begun to take on the true whooping character. There is another thing which you will notice on examining a child which you suspect to have whooping-cough, and that is that he looks puffy about the eyes; the congestion of the head and face which the paroxysm occasions leads to puffiness, which to the experienced eye is often suspicious. You will be apt to mistake for whooping-cough the cough which you get from pressure upon the nerves in the chest caused by *enlarged bronchial glands*. In such a case the history may be of some value, because you know that whooping-cough rarely occurs twice in the same child, and if there is a history of his having had an attack of whooping-cough, and if he develops a similar sort of cough again, the chances are that it is not true pertussis on the second occasion. The other sort of cough which may simulate whooping-cough is that which you get in *bronchiectasis*. But bronchiectasis is not particu-

* 'Brit. Med. Journ.,' August 19th, 1899.

Digitized by Google

larly common in children, and from the clubbing of fingers and signs of cavities in the lungs, and the character of the expectoration, there should be no difficulty in making the diagnosis.

When you have excluded the causes of cough which I have mentioned, those in the lungs and those due to the state of the throat, there remain a considerable number in which you can find no definite cause at all. Some of these are certainly due to gastro-intestinal disorders. There is in children, I think, such a thing as *stomach cough*; at all events you find children who have a constant cough which persists until the state of the digestive organs is attended to. And some people have even described a cough as the result of the presence of worms and the reflex irritation which they produce.

Finally, there is an extremely uncommon form of cough, but one which it is well to be familiar with, and that is the *hysterical cough*. I remember shortly after I started seeing out-patients at the London Hospital, some years ago, a boy being brought who, as he sat in the waiting-room, kept on every half minute or oftener giving out a curious kind of short cough. I examined him without finding any cause for it, and from the history of the case and the general impression one got, I came to the conclusion that it was hysterical. Under the influence of suggestion and one or two doses of valerian the cough stopped absolutely in twenty-four hours. More commonly the hysterical cough is not of that sort, it is of the typical barking character met with in older subjects.

Pyrexia.

The next symptom I wish to consider with you is *pyrexia*, or fever, as an indication of disease in children. I mean by that cases in which pyrexia is the only symptom—where a child is brought to you by the mother because he is hot and feverish, and nothing else has been noticed wrong. These are often cases of great difficulty, and always of very great anxiety in practice, because pyrexia may be, and often is, a sign of serious disease coming on, disease which may be of an infectious nature; and one always wants to make certain at the earliest possible moment what the cause of the fever is. The first thing to look for is signs of the acute *specific fevers*. You should examine the child carefully for one of these, particularly for scarlet fever and measles. In scarlet fever examine the throat for erythema, and look for the early signs of eruption, remembering that it comes out first about the chest and neck. In measles look carefully for catarrhal signs or indications of conjunctivitis and running of the nose. Examine for a rash about the forehead and head. If the rash is not yet out, look for Koplik's spots, which are of very considerable value in the early diagnosis of measles. I need hardly say you should also inquire into the history as regards the possibility of any exposure to infection to one of the acute specific fevers. Failing the discovery of any of those I advise you to look, especially in young infants, for signs of *pneumonia*. I saw in consultation the other day a baby who had suddenly become feverish about three or four days previously. No physical signs were discovered by careful examination, which had been repeated several times after the child was taken ill. When I saw the child there was little to be made out, but at the tip of one lung there was a small patch of tubular breathing and dulness, indicating the development of apical pneumonia. In addition the child had the characteristic grunting pneumonic breathing; and one had no hesitation in saying that the cause of the fever was a small patch of croupous pneumonia at the top of one lung. That is a frequently overlooked cause of pyrexia, as I know from my own experience. I should therefore advise you in any case of acute fever coming on in a young infant to examine the apices of the lungs with particular care, because a patch of pneumonia so small as to be easily overlooked on physical examination may yet be the cause of very high temperature. Another cause of fever which it is important you should remember is *acute gastritis*. I remember two or three years ago seeing a child of about two or three years of age who had been seized with high fever with some abdominal tenderness and vomiting, and the question arose, What was the cause of the symptoms? Various things were suggested, and finally it was thought that the child was suffering from appendicitis, and the possibility of operation was contemplated. But the whole case cleared up entirely after the administration of a few doses of calomel. This was a case of acute gastritis accompanied by high fever. Sometimes, indeed, the fever in such a case is very high, and may be accompanied by rigors. Another disease which you should never forget the possibility of,

Digitized by Google

where fever is present and nothing else, is *otitis media*. Usually in such a case there will be signs of earache in addition. The child will manifest pain or discomfort in the ear; and if you suspect inflammation the best thing you can do is to examine the membranes, and if necessary puncture them, and if you do so you may often find a small quantity of sero-purulent matter escape, with the immediate relief of all the symptoms.

Another cause of acute fever in children which you are apt to overlook is *acute rheumatism* where the joints are not affected. Not long ago a boy was admitted to one of my beds at the London Hospital who had acute fever, and in whom careful physical examination revealed absolutely nothing wrong. But there was one suspicious point, and that was that although he had a temperature of 103°, he was sweating profusely, and further, he looked like a child of the "rheumatic diathesis," as it is called. Acting upon that suspicion we gave him salicylate of soda in full doses. His temperature came down with a run, and a few weeks later he had developed a definite cardiac bruit. This, then, was a case of acute rheumatic fever without any joint manifestations. Such cases are not common, but when they do occur it is very important that you should be able to recognise them, because if you allow rheumatism in a child to go on untreated for a few days, you run a great risk of endocarditis developing and permanent cardiac disease being the result.

There is in young infants a cause of acute fever which you should never overlook, and it is one which is especially apt to escape the observation of a physician, and that is *osteo-myelitis*. I have seen a child lie in the medical ward of a hospital for some days with acute fever, the cause of which was not explained. It died, and post mortem one radius was found entirely surrounded by a layer of pus. That is not unlikely to happen to you in practice, and the moral is, when you have a case of acute fever with no other signs in a young child, you should always examine the bones with great care for signs of tenderness and swelling; always bearing in mind the possibility that the fever may be the result of acute suppurative periostitis or osteo-myelitis. Another point of great importance is, that in every case of acute fever you should examine the *throat* for tonsillitis, which is usually of the follicular sort. I have seen several cases where it was a difficult matter to determine the cause of the fever, but where the whole case was cleared up by looking down the child's throat. It should be done as a matter of routine in every case of disease in children accompanied by high temperature, or in which high temperature is one of the prominent symptoms.

In older children, *enteric fever* will certainly give you a good deal of trouble. I have known several errors in diagnosis made from not recognising the possibility of enteric fever being the cause of the pyrexia. Such a thing happened to me the other day. I had a child in the London Hospital who was admitted with a moderate degree of pyrexia. There were some bronchitic signs in the lungs, rather pronounced, but nothing else. The child looked tuberculous, and one concluded on the whole, seeing that there was no enlargement of spleen, and no spots or anything else, that it was probably tuberculosis, and we were prepared to treat it on that assumption. But as time went on one noticed that the temperature gradually mounted up, and then the spleen began to be palpable. The Widal reaction was tried, and definite clumping occurred at once. That of course made the diagnosis clear. Now, such a mistake as that is very easy to fall into. The recognition of enteric fever in young children is more difficult than it is in grown-up patients, and if you have fever for which you can find no other cause, even in the absence of enlargement of the spleen or other signs, bear in mind the possibility that you may have to do with a case of enteric fever, and, by having the Widal reaction done, or by estimating the number of leucocytes, try to arrive at an exact diagnosis.

There is a cause of fever in infancy which is of rare occurrence, but which it is extremely important that you should recognise when it does occur, and that is *acute pyelitis*. Last summer I was called in consultation to see a baby who presented some rather puzzling features. She was a girl of a few months old who had for some weeks been running a very high temperature, with occasional remissions, and—this is one of the suspicious and important points—accompanied at times by definite rigors. On inquiry into the state of the urine it was found to contain pus. One then felt certain that it was a case of pyelitis. Further examination showed that the urine contained the

Digitized by Google

Bacillus coli communis, and on giving the child alkalies in full doses, which is the right treatment in such cases, the temperature speedily came down.

Inability to Talk.

Passing from pyrexia, I want to take up together two symptoms which mothers frequently complain of in their infants. One of these is *inability to talk* at the proper age, and the other is *inability to walk*. If a child is brought to you because of inability to talk, you should bear in mind that there are only a few possibilities which you need consider. In the first place of course the child may be a *deaf mute*. In such a case you will find quite a clear history that the child is absolutely deaf, and you will sometimes find that there is a history of several similar cases having occurred in the same family. Such cases should not present any great difficulty in the matter of diagnosis. You find, again, that some children are unable to talk at the proper time because of *mental deficiency*, or else because of backwardness short of actual mental deficiency. These cases are more difficult to recognise. You should examine the child for the physical stigmata of mental deficiency, for cranial abnormalities, and the other signs which you know to be the outward expression of retarded development of the brain. But in the lesser degrees of mental deficiency, where such physical signs are absent or are not pronounced, you may often be in doubt as to whether that is truly the cause of the child being backward in talking.

Pathological conditions in the throat, especially *adenoids* and *enlarged tonsils*, may be a cause of difficulty in learning to talk; I cannot exaggerate to you the importance of this fact. I should say that the great majority of children I have seen who were brought to me because they were unable to talk at the proper age were suffering from some abnormality of the throat. A typical case of this kind was that of a girl two years and three months old, who was brought by her mother because she was unable to talk, although "she made violent efforts to do so." The mother said the child heard well, she was obviously quite intelligent, and the only thing one could make out on physical examination was the presence of an enormous mass of adenoids in the naso-pharynx. These were removed by operation, and five months later the mother wrote that the child was talking well. That is a clear and typical instance of what I am referring to, and I could quote dozens of such cases almost which I have seen in the out-patient room here and elsewhere. It is difficult to say in such a case why it is that adenoids make it difficult for the child to learn to talk. The mother is probably correct when she says that the child does hear, but the possibility is that he does not hear quite distinctly, not well enough at all events for him to learn to imitate the speech of other people. Whether it is that, or whether the adenoids impose a mechanical obstacle to movements of the soft palate I do not know, but I know if a child is brought to you with the complaint that he cannot talk, and you find adenoids present in large amount, you can promise with almost complete confidence that if the adenoids are removed the speech will become perfectly natural.

A rare cause of this lack of the ability to speak is *tongue-tie*. Mothers think it is a common affection, and most children who have difficulty in learning to talk are popularly believed to suffer from it. Now, tongue-tie does occur, but it is very rare. I can only recall at this moment three well-marked cases of it, and I doubt if it ever does produce true inability to talk; that is to say, real dumbness. It may cause difficulty in talking clearly and in pronouncing certain consonants for which the tongue is particularly required, but you must distinguish that from inability to talk at all. A still rarer cause of inability to talk is *congenital aphasia*, a condition of which I have never met with an example, but which is described as one of the occasional congenital defects of childhood.

Inability to Walk.

Now to turn to the kindred symptom, inability to walk. There you have to recognise, I think, two groups of cases; first, those in which the child has never walked at all from birth; and, second, those in which the child has at one time or another been able to walk, but has "gone off his legs," as the mother puts it.

First, with regard to those who have never walked. There you have three possibilities to deal with; either the child is the subject of rickets or of some form of paralysis, or it is a case of mental deficiency. Now, in *rickets* the difficulty is merely one of walking. Whilst the child sits in his mother's lap he can move his legs about freely,

Digitized by Google

whereas in cases of paralysis the child is not only unable to walk, but he cannot move the affected limbs, even when sitting. In rickets you will find also the other general signs of the disorder—enlargement of the epiphyses, deformity of the chest, and so on,—the knee-jerks also are unaffected, whereas in paralysis they are exaggerated or diminished.

Of *paralysis* as a cause of inability to walk there are two chief varieties: one is infantile paralysis, the other is spastic or cerebral paralysis. Infantile paralysis usually comes on definitely at some period subsequent to birth, whereas spastic paralysis is often congenital. In infantile paralysis you will find the knee-jerk on the affected side diminished and the muscles atrophied. In spastic paralysis you will find the knee-jerk exaggerated and the muscles of the limb well developed.

Mental deficiency you will recognise by the other general signs of mental impairment which I have mentioned before. Never forget, however, the possibility that children may be unable to walk from surgical reasons as well as medical. I have seen cases of *congenital double dislocation* of the hip produce great delay in learning to walk, and such cases are apt to be overlooked by the physician whose mind is fixed upon medical points.

When a child who has hitherto been able to walk "goes off his legs" you are dealing with a different order of cases. Many children who have had an acute illness will go off their legs for a long time after that illness, although you can find no special cause for their doing so. Probably it is partly because of muscular weakness, and partly because the child actually forgets how to walk—having only recently acquired the rather difficult art of walking, he gets out of practice when laid up in bed during an acute illness, and when he gets on to his feet once more he has to learn all over again. Such cases may go on without walking for a long time; but you may rest assured that they will walk in time. Another cause of a child going off his legs is that he may have developed infantile paralysis, but I have already told you how that is to be recognised.

A rarer cause which it is important to recognise is *rheumatism*, slight degrees of which affecting the hip or the muscles at the back of the knee-joint or ankle may cause the child to go off his legs on account of pain.

Remember again the possibility of *surgical causes*. One of the earliest signs of hip disease may be that the child goes off his feet.

Paralysis.

A considerable number of children will be brought to you by the mother with the complaint that they suffer from "paralysis." We must therefore consider the possible causes of paralysis as a symptom in childhood. The first thing I should advise you to do here is to try to determine whether you are dealing with true paralysis, by which, of course, I mean an affection of muscles or nerves, or merely with what one may describe as a *pseudo-paralysis*, for a number of children who for any reason may have pain in a limb may cease to use that limb, not because they have lost control over its muscles, but because any movement of it causes them great discomfort. There are only three conditions which are likely to produce pseudo-paralysis. The first of them, and the best marked, is *congenital specific epiphysitis*. I have seen great difficulty experienced over these cases, and many errors of diagnosis made with regard to them. Not long ago a child was admitted under my care at the London Hospital, who had suffered for a short time from what was described as "paralysis of one arm." The condition had followed some trivial accident, and it was believed by the mother that the child's arm had been injured, and that paralysis had resulted.

On examination there was apparently very little to be made out, but it was obvious that in the first place the child allowed the arm to hang limply by his side, and that passive movement at the shoulder produced pain, whilst there was apparently some thickening about the neck of the humerus. On other grounds than those of the local condition one concluded that the child was the subject of congenital specific disease, and treatment by grey powder was vigorously carried out, with the result that in a short time the child became well. This was a case of specific epiphysitis involving the humerus and simulating paralysis, and it is usually for the apparent paralysis that such cases are brought for advice. You should remember that acute specific epiphysitis usually affects more than one joint, and secondly, that it always occurs in quite early infancy. The majority of cases occur only a few weeks after birth, and I never saw a child affected in that way who was older than six

Digitized by Google

months. You need not consider the possibility of this disease being present in a child who is over six months old. After six months old you meet with *scurvy* as a cause of pseudo-paralysis. Two or three weeks ago a child was brought to the receiving room of the London Hospital, who had lost the use of one leg. The case was sent down to out-patients with the diagnosis, "paralysis, ? cause." On going into it one found in the first place that any attempt to move the leg produced severe pain. There was no alteration of the reflexes or sign of wasting or any other indication of a true paralysis, but the gums were spongy, and the diet had been of a sort calculated to produce scurvy. The child was put on an antiscorbutic diet, and in a week's time he was moving the limb freely. That is a typical example of scurvy simulating paralysis. The third cause of pseudo-paralysis in children is *rheumatism*, but I have referred to this as a cause of a child going off its legs, so I need not say any more about it now.

Turning now to the causes of *true paralysis* in childhood, there are only two common forms which you need consider. One of these is infantile paralysis, and the other is cerebral, or spastic, paralysis. The spasticity of the one and the limpness of the other will prevent your having any great difficulty in deciding to which any given case belongs.

Headache.

Another nervous symptom which children present, and which may often lead to considerable difficulty in diagnosis, is headache. Now, the first thing one has to remember about headache in children is this, that the causes of it are usually totally different from those in grown-up people. One of the commonest causes of headache in grown-up people is some vascular disturbance, some cerebral congestion, or cerebral anæmia, or high tension in the intra-cranial vessels; but these are not common causes in children. On the other hand, what one may call "reflex" causes of headache are common in childhood, and particularly eye-strain. If a child of school age suffers from headache, I should recommend you to examine his refraction and see if there is any hypermetropia or astigmatism, and if you find either, to get the error corrected by proper glasses. If you do that you will often find that the headache disappears at once. Another group of causes of headache may be described as *toxic*, constipation and biliousness are examples, just as in grown-up people, and these are to be met by appropriate remedies. *Cerebral tumour*, again, occurs with considerable frequency in children, and may be a cause of severe headache. Here you should inquire for other symptoms of tumour, such as vomiting, and look particularly for optic neuritis. *Adenoids* are a pretty common cause of headache in childhood. Here the headache is usually frontal. *Migraine* also is a disease of comparative frequency in children, in whom it occurs quite as often as in grown-up people, and frequently with great severity. Hysterical headache, on the other hand, is a condition of very rare occurrence in early life.

Hæmaturia.

A minor symptom which it is important that you should be familiar with, as it often gives rise to difficulty in diagnosis, is hæmaturia. A child may be brought to you by the mother with the statement that he passes blood in the urine. There is here one fallacy which I would warn you against, and it is that mothers are apt to regard as blood what is really not blood at all, but a brick-dust deposit of urates. But assuming you have verified the fact that the child is passing blood, there are but few possible causes of it which you have to consider; indeed, I think there are but four causes of hæmaturia in childhood which it is necessary for you to bear in mind. One is *hæmophilia*, which may be a cause of hæmaturia, especially in boys. Another cause is *scurvy*, which I believe is commoner than one is disposed to think, and is often overlooked because examination of the urine of young infants is apt to be omitted. I remember a child who was under the care of a surgeon in a hospital for the passage of blood, and who was believed to be the subject of stone in the kidney. An operation was performed, but no stone was found. A physician then saw the child and inquired into the diet; the replies given led him to think that it was a case of scurvy, and on putting the child on to fresh vegetables and fruit the hæmaturia disappeared in a few days. This, then, was a case in which failure to remember that scurvy may be a cause of hæmaturia led to the performance of an entirely unnecessary operation. The third cause of hæmaturia is the *passage of urates*

Digitized by Google

and oxalates. The hæmaturia in such a case is usually, I think, paroxysmal or intermittent, coming on and lasting for a few days and then passing off again. Careful examination will show the presence of crystals of uric acid or oxalic acid; and suitable treatment will cause the blood to disappear. The fourth cause is *sarcoma of the kidney*, and one which it is well to bear in mind. Not long ago I saw a case where this gave rise to great difficulty. It was that of a young child who passed a trivial amount of blood in the urine, but passed it continually. Oxalates were discovered in the urine, and it was thought that they were the cause of the hæmaturia. But special treatment directed to remedying this did not affect the passage of blood, although the oxalates ceased to appear. By-and-by an abdominal tumour became evident, and operation showed it to be a renal sarcoma. Remember always, then, that continuous small bleedings may be a comparatively early sign of sarcoma of the kidney in a young child.

Croupy Respiration.

A symptom of very great importance, and one which produces great anxiety in the minds of the friends, is that spoken of as laryngeal or croupy breathing. You are called to a child who exhibits croupy breathing, and you have to determine what the cause of it is. I need hardly say at the outset that the two things you will first think of, quite rightly, are so-called "croup" on the one hand, and diphtheria on the other. Now, "croup" usually means a catarrhal laryngitis, or it ought to mean that. Of this there are two sorts: first, that in which there is a slight degree of catarrh, but every now and then there is spasm superadded, producing croupy breathing. Second, there are the more severe cases, in which the catarrh of the larynx is so great that it causes considerable swelling of the mucous membrane and produces more or less continuous obstruction. The first sort of case you are not likely to confuse with diphtheria, because the history is that such a child is comparatively well during the day, but that during the night attacks of croupy breathing come on. The real difficulty arises in the second group of cases, in which the catarrh has led to so much swelling as to produce continuous obstruction. These are the cases in which it is difficult to tell whether the case is one of diphtheria or not. In some cases, indeed, the diagnosis may be impossible. But there are certain points to which you should direct your attention. First, there is the mode of onset in the two cases. In catarrhal laryngitis the onset is usually more abrupt and severe than in diphtheria, which begins more insidiously. Catarrhal laryngitis is accompanied by a higher degree of fever. In the second place, the loss of voice and cough are less in catarrhal laryngitis than in diphtheria. In other words, diphtheria is the more *silent* disease of the two. If you find signs of a membrane on the fauces of course you need have no difficulty at all, but the puzzling cases are those in which the diphtheritic process is confined to the larynx only, and although the above indications may be of some help, every now and then you are bound to be left in doubt. In such a case the wisest plan is to act as if it were the more serious disease; that is to say, isolate the patient and give antitoxin.

But there are other causes of croupy or laryngeal breathing which it is well to bear in mind. It may occur, for instance, as a result of *retro-pharyngeal abscess*, and I have known serious mistakes arise in this way. If in doubt, put your finger into the child's throat and explore the pharynx and see if you can find any indication of bulging. Croupy breathing may also occur in the course of a *chronic laryngitis*, which may be simply catarrhal or the result of congenital syphilis. It may occur also from pressure on the trachea, by *enlarged bronchial glands*, or, in rare instances, by an *enlarged thymus*. It may also be simulated by certain conditions, the most notable of which are *congenital laryngeal stridor* and *laryngismus stridulus*, although there the nature of the attacks should leave you no doubt. Congenital laryngeal stridor dates from birth, whilst laryngismus stridulus is always preceded by a period of apnœa, followed by a long crowing inspiration, which does not resemble the continuous croupy breathing of laryngeal obstruction It is always well also to remember the possibility that you may be dealing with a *papilloma of the larynx*. Two or three years ago a baby was brought here in my absence and seen by my assistant. The child had continuous croupy breathing, and the question arose, what was the cause of it? The symptom had been noticed since birth, and it was rightly thought that it might be a case of congenital laryngeal stridor. The child died suddenly within a week, and the post-mortem

Digitized by Google

examination revealed the presence of laryngeal papilloma. I have since seen two cases where there was continuous slight croupiness of breathing, and in which removal of a papilloma caused the disappearance of the croup. Lastly, bear in mind always the possibility that croupy breathing may be the result of inhaling a *foreign body*. Careful attention to the history will generally keep you right here, because the onset in such a case is always, of course, extremely sudden.

I had intended to consider with you some other symptoms, such as screaming, enlargement of the abdomen, convulsions, sleeplessness, and disturbed nights, but these I must reserve for a future lecture.

Queen Anne Street, W.;
March 15th, 1904.

Fasting in Treatment.—Sands finds that prolonged fasting is a very excellent treatment, according to his experience, in typhoid fever, and that patients suffering from pneumonia and influenza, as well as after severe injuries and after operations, also do better without nourishment for a while. The most convincing results, however, are in the gastro-intestinal disorders, such as typhoid. It is his rule when called to see a typhoid case to tell the family frankly that the patient should be kept on a strict water diet from eighteen to twenty-one days, assuring them of a more comfortable illness and more certain recovery. He finds that this plan relieves the abdominal symptoms, keeps the temperature lower, and is otherwise generally beneficial. He summarises as follows:—(1) Acute illnesses in general, of any severity, do better without nourishment, for the reason that, on account of the fever and the poison of the disease, the food is not digested or assimilated, and, if not, adds a gastro-intestinal disturbance to the already existing disease. (2) In acute diseases of the alimentary canal in particular all nourishment should be withheld until complete subsidence of symptoms. It is quite possible for a patient to fast twenty-one days, or even longer, without the development of any alarming weakness. (3) The indications for the allowance of food are the absence of abdominal symptoms, a cleaning of the tongue, and a continuously normal or subnormal temperature.—*Journ. A. M. A.*, vol. xlii, No. 9.

FOUR LECTURES
ON
NORMAL AND ABNORMAL CONDITIONS OF THE BLOOD.*

By EDMUND CAUTLEY, M.D.Cantab., F.R.C.P.Lond.,
Physician to the Belgrave Hospital for Children and the Metropolitan Hospital.

LECTURE II.
PRIMARY AND SECONDARY ANÆMIAS.

GENTLEMEN,—The division of cases of anæmia into two groups, according to the presence or absence of splenic enlargement, has proved unsatisfactory for several reasons. Thus, enlargement of the spleen may be due to the anæmia or to the underlying cause of the anæmia, most probably the latter. In size it bears no definite relation to the degree of anæmia. Grave anæmia may occur without any increase in the size of the spleen. And enlargement of the spleen may be found in inherited syphilis, and even in rickets, without there being any anæmia. A palpable spleen is not necessarily an enlarged one. In infants the spleen can be easily felt, even when not enlarged.

For practical purposes we may divide anæmia into three groups of cases:

1. PRIMARY ANÆMIAS—cases in which no cause is found, or in which any cause present is not deemed sufficient to account for the degree of anæmia. Under this heading are included pernicious anæmia and the various types of true leukæmia.

2. SECONDARY ANÆMIAS—cases in which a definite cause is present, and is deemed sufficient to account for the degree of anæmia. These are sub-divided into those with and those without leucocytosis, either of which may be mild or grave.

3. INTERMEDIATE ANÆMIAS—a group of cases which are with difficulty differentiated from the other two groups. This includes anæmia splenica infantum, anæmia infantum pseudo-leukæmica or Von Jaksch's disease, Hodgkins' disease, lymphadenoma, lymphatic anæmia, and the splenic anæmia of Banti.

The question arises, and will be discussed sub-

* Delivered at the Medical Graduates' College and Polyclinic.

Digitized by Google

sequently, whether all the anæmias of infancy are of the same type, and differ only in degree. For the present we will adopt the above classification, and deal first with cases of secondary anæmia.

Secondary Anæmia.

Mild cases of secondary anæmia are those in which the blood shows no deformity of the erythrocytes, and may or may not be deficient in hæmoglobin. They are more often than not associated with leucocytosis. Grave cases exhibit deficiency in the hæmoglobin, degeneration of erythrocytes, poikilocytosis, and usually leucocytosis. Probably there are various stages, and one form may pass into another. It is even possible that a severe anæmia, with or without leucocytosis, in an infant may develop into a variety of intermediate anæmia, or even into true leukæmia or pernicious anæmia.

Etiologically any disease may produce a secondary anæmia through an impairment of the function of blood formation, or an increased destruction of blood, or by a combination of the two processes. Some cases are congenital in origin, and result from a morbid condition affecting the mother during pregnancy, such as syphilis, tuberculosis, malaria, or merely general delicacy and malnutrition. Premature infants readily become anæmic. More often the anæmia is acquired as a result of improper feeding, a prolonged diet of cooked food, gastro-enteric derangements, affections of the kidney, suppurations, febrile conditions, blood diseases, rickets, and tuberculosis. Or, it may result more directly from actual hæmorrhage from the nose, umbilicus, bowels, etc., or from hæmorrhagic affections such as scurvy, purpura, and hæmophilia. Intestinal toxins passing into the blood and escaping destruction in the liver may produce similar results by direct action on the blood-forming organs, on the marrow of bones, or on the blood itself.

By *examination of the blood* we can differentiate cases according to the degree of severity. In mild anæmia there is little reduction in the number of red cells, the percentage of hæmoglobin, and the specific gravity. In the severe forms there is considerable reduction in all these factors, with poikilocytes and hæmatoblasts in varying degree. Leucocytosis may or may not be present. Cases in which it is present are more common and more severe. The spleen is almost invariably enlarged, except in cases which are mild and exhibit no leucocytosis. Occasionally very severe anæmia is seen in infants without any splenic enlargement. Put shortly, the more severe the anæmia the greater is the reduction in the specific gravity (down to 1035); the more marked is the reduction in the number of red cells (down to two millions), and the percentage of hæmoglobin (down to 30 per cent.); the more marked is the poikilocytosis; the greater is the number of normoblasts and megaloblasts; and the greater is the leucocytosis (up to 65,000).

The *symptoms* are similar to those we find in adults, such as pallor, fretfulness and irritability, shortness of breath worse on exertion, digestive disturbance, constipation, headache, insomnia, and muscular weakness.

The *diagnosis* depends upon the recognition of the cause and the characteristics of the blood. The latter are of less value in infants than in older children, for a leucocytosis is so easily developed in infancy. The presence of splenic hyperplasia is common in all forms of anæmia, except pernicious anæmia, and suggests other diagnoses, such as leukæmia, or one of the varieties of intermediate anæmia. These can only be excluded by the results of a careful blood examination, and not always then.

The *prognosis* depends upon the cause, the age, the extent of the blood changes, and the general condition of the patient. Those cases due to simple hæmorrhage are easily recovered from. Those resulting from errors in diet, bad hygienic conditions, and gastro-intestinal causes, will be the more serious in proportion to the blood changes and to the possibility of removing the cause. In cases due to toxæmic conditions, such as diphtheria, or to prolonged loss of albumen, such as from chronic nephritis or empyema, the prognosis is that of the original disease. Usually death results from an intercurrent disease. In a few instances the disease has progressed on into leukæmia or pernicious anæmia, or has been a mild form of one or the other of these conditions from the start. Practically the greater the anæmia, as judged by the examination of the blood, the worse is the prognosis.

The following case is illustrative of the extreme difficulty we may find in making a diagnosis and in

Digitized by Google

strictly differentiating the varieties of anæmia and splenic hyperplasia according to the classification I have given you.

A boy of fifteen months died under my care after an illness characterised by left facial paralysis and otorrhœa, enlarged cervical glands, most marked on the left side, hard, movable, separable, painless, and non-adherent to the skin; dulness over the manubrium and in the right first intercostal space; spleen extending down to the level of iliac crest; liver three inches below costal margin. Blood examination: white cells, 60,000; red cells, 7,470,000. Small lymphocytes, 14·4; large lymphocytes, 31·5; polynuclear cells, 53·9; eosinophiles, a few. He developed progressive consolidation of the right lung, and a febrile condition suggestive of tuberculosis or septic infection.

Post-mortem.—The glands in neck, mediastinum and mesentery in appearance suggested Hodgkins' disease. In neither liver, spleen, nor lungs was there any evidence of localised or generalised leucocytic infiltration, but numerous large giant-cells were found. Possibly the case was one of lymphadenoma with secondary tuberculosis and septic infection. (For fuller details *vide* 'Reports of The Society for the Study of Disease in Children,' vol. iii, p. 11, 1903.)

Pernicious Anæmia.

This is a variety of primary anæmia which exhibits definite blood changes, progresses rapidly, and almost invariably ends fatally. Possibly it may be subdivided into splenic and hæmolymphatic types, the latter arising in the hæmolymph glands. It must be carefully distinguished from fatal cases of secondary anæmia. An anæmia is not necessarily a pernicious anæmia because it is fatal. The cause is not absolutely determined. One or more of all the factors giving rise to secondary anæmia may be in action, but a further factor is said to be necessary. William Hunter has brought forward strong evidence that this further factor is some kind of toxic poison which causes hæmolysis, and that this poison is formed in the mouth or alimentary canal. He holds the view that the disease is caused by a septic infection, due to the *Streptococcus pyogenes longus*, *plus* a specific hæmolytic infection. Cases have been found associated with intestinal parasites, such as tape-worms and the anchylostoma, or with diseases such as inherited syphilis, rickets, and especially splenic enlargement in infants. These latter cases suggest that the affection in infancy may be a further stage of secondary anæmia.

Morbid anatomy.—The skin is lemon-yellow in colour. The body is usually well nourished, but may be emaciated. All the organs are excessively anæmic. The heart, liver, kidneys, and pancreas show fatty degeneration. Iron is deposited in the liver tissue and the mucous membrane of the intestines, and is in excess in the kidneys and spleen (hæmosiderosis). The spleen is hard and may be a little enlarged from fibrous hyperplasia. Capillary hæmorrhages are found in the skin and internal organs. The bone marrow shows the most typical lesions, and is embryonic in type, containing many normoblasts and megaloblasts. According to Warthin the prevertebral lymph glands, especially the cervical, thoracic, and retro-peritoneal, are affected. Glossitis, septic conditions of the mouth, septic gastritis, and a croupous form of enteritis, have at times been found.

The *blood* is watery, dirty-pink, reddish-brown, or coffee-coloured. It possesses feeble power of coagulation and does not form rouleaux. The red cells are diminished down to 800,000 to 2,000,000, and sometimes even to 500,000. The hæmoglobin is reduced to fifteen to twenty-five per cent. The colour index may be high, that is, there is a relatively high percentage of hæmoglobin in the individual erythrocytes. The other changes include marked poikilocytosis and polychromatophilia; a preponderance of megalocytes; the presence of "shadow corpuscles" and microcytes; an increased number of megaloblasts and normoblasts, especially the former; karyokinesis of the nuclei of red cells; sometimes a few myelocytes; a normal number or deficiency of leucocytes, occasionally an excess; and a relative lymphocytosis in bad cases. Most of the few cases in infants have shown a moderate leucocytosis. The blood has not the same diagnostic value in infants as in older children and adults, because of the instability of the blood-making function. Thus megaloblasts, normoblasts, macrocytes, and poikilocytes may occur in grave anæmia which cannot be classified as pernicious. The hæmolysis differs only in degree from that occurring normally in certain diseased conditions.

The *symptoms* and *course* are the same as in

Digitized by Google

adults. The disease is rare in infancy and uncommon in childhood. Audeoud collected twenty-five cases, of which only four were under two years of age. One case is reported at three months of age. An insidious onset is followed by the development of profound anæmia and prostration, without marked emaciation. Hæmorrhages take place *sub cutem*, into the retina, or from the nose, stomach, bowels, lungs, etc.

A special form of glossitis has been described by Hunter as being almost constant in the early stages or during the course of the disease. The tongue becomes tender, sore, and swollen, with little vesicles forming on the surface. The attacks are recurrent. In the late stages the tongue may be normal. He has also found septic infection of the mouth, septic gastritis and enteritis. The pulse is full and soft. Hæmic murmurs, cardiac dilatation, and œdema are common. The urine is scanty, pale, and of low specific gravity. Both spleen and liver may be enlarged. The temperature is variable, and may remain at about the normal level.

The *diagnosis* depends upon the results of the blood examination, taken in conjunction with the symptoms and progressive course. The *prognosis* is almost hopeless, and the disease usually ends fatally in a few months to a year. To a certain extent it is dependent upon the relative number of hæmatoblasts found. Thus, it is worse when the megaloblasts are in excess, and better if the normoblasts are more numerous or, on frequent examination, are evidently increasing in number. The cure of any predisposing or exciting conditions of anæmia improves the prognosis.

Leukæmia.

Leukæmia is a special form of anæmia in which certain very characteristic changes are found in the blood. The essential features are an increase in the number of white cells, with a relative alteration in the percentages of the different varieties, and changes in the spleen, lymph glands, and bone marrow. It is not a simple leucocytosis. Abnormal cells may be present. It is the result of the manifestation of some poison which affects the bone marrow especially, and ultimately causes death. The etiology is obscure. Many cases are apparently primary, while others appear to be secondary, a further development in the course of an anæmia belonging to the group of intermediate or secondary anæmias. The possibility of an infectious origin in a few cases must be borne in mind. The disease is rare in infants and young children. Four congenital cases have been recorded. One of these, recorded by Pollman in 1898, is interesting as showing the possibility of an infectious origin.

The child died nineteen days after birth. Post mortem, patency of the ductus arteriosus and ventricular septum, and endocarditis of the tricuspid valve were found. The blood showed all the characteristic features of acute lymphocytic leukæmia. Possibly this was merely an exaggeration of the lymphocytosis of the new-born, due to septic infection.

Of cases recorded under two years of age, an element of doubt enters into the diagnosis. They are usually described as such on insufficient evidence. In Stilling's case (1880), no blood-count was made during life. The child died at twelve months of age, and showed leukæmic changes post mortem. Von Jaksch (1889) recorded a case of a child of fourteen months with a leucocytosis increasing up to 192,000, and typical leukæmic changes post mortem. No differential count was made. This appears to be an undoubted case. Middleton recorded in 1893 the case of a child of sixteen months with a leucocytosis of 370,000. Post mortem, the capillaries were found stuffed with leucocytes. This might be an intermediate case. The one recorded by Morse in 1894 is still more doubtful. The leucocytosis only amounted to 48,000, a number which may be readily reached in an intermediate or secondary anæmia. No post-mortem examination was made. Thus in all these cases the blood when examined hardly presented the changes we should expect to find in true leukæmia in infancy. Considering the readiness with which a leucocytosis is produced in infancy, one would expect to get a much higher proportion than even the 192,000 found in Von Jaksch's case. In older children, from a half to one million white cells are found. We ought to get at least as high a proportion in true leukæmia in infancy. McCaw ('Reps. Soc. for Dis. of Children,' vol. iii., 1903) has reported a case which was apparently one of true leukæmia in an infant æt. twenty months. The white cells amounted to 810,000, and 99·2 per cent. of them were lymphocytes. A few hæmato-

Digitized by Google

blasts were found. The red cells were reduced to 2,200,000, and the hæmoglobin to 39 per cent. The spleen, liver and glands were enlarged; the spleen extending down to the brim of the pelvis. The child presented many of the clinical symptoms of leukæmia and died. Unfortunately no autopsy could be obtained.

Varieties.—Enlargement of the spleen is not an essential feature, and probably neither the spleen nor the glands are primarily involved. Three types have been recognised, of which one, the purely myelogenous, is unknown in infancy. The diagnosis of the different types is partly anatomical, depending on the organs specially involved; partly clinical, depending on the variety of white cell prominent in the blood; and partly pathological, depending on the nature of the change causing these conditions. We are justified in dividing the cases into two groups: (1) the spleno-myelogenous, occurring primarily in the marrow; (2) the lymphocytic or lymphatic, occurring primarily in the lymph glands.

Acute leukæmia is almost invariably lymphocytic. Hirschfeld and Cabot have recorded cases in which it was myelogenous. According to some writers (Brandenburg, Pappenheim, Walz), in every case the bone marrow is the primary seat, and the other organs are secondarily involved, the blood not becoming leukæmic in the lymphocytic type until the marrow is involved.

Acute leukæmia may show no enlargement of the spleen or glands, but exhibits lymphadenoid degeneration of the marrow, a reversion to the fœtal condition, and intense lymphocytosis in the blood. Lymphoid cells, identical with lymphocytes, are always present in the marrow in considerable number, and probably by their proliferation make their way into the blood. Morphologically, the two are identical. The lymphocytosis, therefore, may be due to proliferation of the lymphoid cells in the marrow. On the other hand, it is possible that the lymphocytes in the blood are derived mainly from lymphoid tissues throughout the body generally. Against this it may be pointed out that lymphocytosis is absent in Hodgkins' disease and other affections of the glands. Possibly both types of leukæmia arise from toxic or infective causes which, by a process of chemiotaxis, act on different cells in one individual to what they attack in another.

In the lymphocytic type we find general enlargement of the lymph glands and lymphoid tissues, or of the latter only, the enlargement being due to a simple hyperplasia. The spleen and liver are moderately enlarged. This is the most common type in infants and young children. In the spleno-myelogenous type the spleen is usually very much enlarged from simple hyperplasia, and contains an excess of lymphoid tissue. The liver is also enlarged and contains an excess of lymphoid tissue. The bone-marrow shows hyperplasia, and in it are found nucleated red cells in all stages of development, myelocytes, and all sorts of abnormal red cells. The glands are little or not at all enlarged, with the exception of the mesenteric glands.

Examination of the blood.—The percentage of hæmoglobin is reduced to 25 to 50. Except in the early stages, the number of red cells is reduced to about half the normal, and a variable degree of poikilocytosis is present. The leucocytosis is the great feature. The proportion of white to red cells may be increased to one white to three red cells. It is most marked in the spleno-myelogenous type. In the lymphocytic form there is an extreme lymphocytosis (up to 97 per cent.), and a diminution in the actual and relative numbers of the other white cells. In the spleno-myelogenous type there is a great relative deficiency of lymphocytes. Other changes are shown in the accompanying table:

Blood in Leukæmia.

	Lymphocytic. A pure lymphocytosis.	*Spleno-myelogenous.* A myelocytosis.
Lymphocytes ...	96	10—30
Polynuclears ...	3	50
Eosinophiles ...	Rare	5
Myelocytes ...	Few or absent	35—60
Hæmatoblasts ...	Rare	Very numerous.

The above table represents the results found in adults and older children. No complete data as to the blood in the leukæmia of infancy are available. A total leucocytosis is not necessarily present throughout the course of the disease, but the relative changes probably persist. In fact the qualitative alteration is the feature of importance, and differential staining becomes essential for diagnostic purposes. The blood changes do not always correspond, for Rose Bradford has found

Digitized by Google

in adults that spleno-medullary blood may be associated with lymphocytic leukæmia. The presence of many "mast-cells" is regarded by Ewing as pathognomic of the spleno-myelogenous type. Eosinophiles and myelocytes appear with great readiness in the blood of infants, so we should expect a great increase in true leukæmia in infancy.

Morbid anatomy.—The marrow, especially that of the long bones, is affected in all cases; the fatty marrow is replaced by "red currant jelly" marrow or more rarely by a puriform gelatinous material. The thymus is always persistent or enlarged, and not infrequently forms a definite lymphatic growth. The mesenteric glands are always enlarged, and the spleen may be enormous. The liver is frequently enlarged, especially in the spleno-medullary type, and may be enormous. The accessible glands are enlarged in about one third of the cases and are softer than in Hodgkins' disease. The blood resembles *café au lait.* Large clots are found in the heart. Petechial hæmorrhages are common in serous membranes, especially in the pericardium and pleura. Occasionally intramuscular hæmorrhages occur. Sometimes there is an extraordinary deposit of uric acid encrusting the mucous membrane of the pelvis, of the kidneys and ureters.

The *onset* is insidious, with progressive weakness and increasing pallor. Some patients may have quite a good colour in the early stages. The abdomen enlarges from increase in the size of the spleen and liver. This and the anæmia cause shortness of breath. Dyspnœa may be paroxysmal and mistaken for asthma. It is usually due to exertion. General glandular enlargement occurs in the lymphocytic type, and sometimes to a slight extent in the other forms. The other symptoms include dyspepsia and attacks of diarrhœa; fainting attacks; hæmorrhages from mucous membranes and subcutaneously, the bleeding being most common from the gums, nose, stomach, and bowels, and occasionally from the lungs and kidneys; œdema of the feet and general anasarca in late stages; frequent, soft pulse; moderate and irregular pyrexia; albumen and casts in the urine; and occasionally attacks of colic due to the deposit of uric acid in the kidneys.

Fourteen cases of acute leukæmia have been recorded. R. Dods Brown (1903) reported a case in a boy æt. 13 years. The symptoms were bleeding from the nose, gums and throat; hæmatemesis and melæna. At the autopsy there were found marked hæmorrhages throughout the body and a much enlarged thymus, crowded with lymphocytes. Bradford and Shaw reported one in a boy æt. 7, and Macrae one in a boy æt. 3. The disease is of sudden onset and simulates some varieties of purpura, and ends fatally in a few days to about eight weeks. It is characterised by anæmia; fever; purpuric rash; hæmorrhages from nose, mouth, stomach, bowels, or kidneys; affections of the mouth and tonsils with enlargement of the cervical glands in about half the cases; general glandular enlargement in a smaller number. The spleen is always enlarged, and the liver is enlarged in more than half. The gums may be swollen, dusky, and ulcerated. The blood-picture is that of lymphocytic leukæmia, and the diagnosis depends on the blood examination. Both the red cells and specific gravity of the blood are reduced.

Diagnosis.—The differentiation of the different types depends mainly on the blood examination. In the lymphocytic type the glands are usually more and the spleen less enlarged than in the spleno-medullary type. From other diseases it is diagnosed by the presence of abnormal cells (myelocytes and hæmatoblasts) in one type, and upon a marked lymphocytosis in the other. In the latter case the relative alteration is the important change, taken in conjunction with the age of the infant or child. It is very important not to confound a secondary anæmia with leucocytosis and splenic enlargement, so common in infants, with true leukæmia. The disease must also be diagnosed from the different varieties of intermediate leukæmia, more especially Hodgkins' disease, anæmia infantum pseudo-leukæmica, and chronic adenitis. The presence of myelocytes is not conclusive; they may be found in pernicious anæmia, but not in abundance or in association with leucocytosis. They may occasionally be found even in large numbers in the course of anæmia infantum pseudo-leukæmica. Such a case came under my care in a boy æt. 13 months. At one period he lost weight rapidly, developed numerous petechial hæmorrhages, and his blood contained many myelocytes. In spite of this he slowly recovered.

It is quite possible that Hodgkins' disease may terminate in acute lymphocytic leukæmia. Such a case was recorded by Wende ('Amer. Journ. Med.

Digitized by Google

Sci.,' Dec., 1901) in a man æt. 26. At first the blood-count was normal. The patient improved for six weeks under treatment, then suddenly got worse and died in three weeks. The blood contained 95·5 per cent. of small lymphocytes and mixed changes were found post mortem. If such a result can occur in an adult it is still more likely to take place in infants, seeing that the blood reverts so readily to the lymphocytic type.

The *prognosis* is bad. Except for the few acute cases the disease is chronic and progressive. It is worse both as regards duration and issue in the lymphocytic type than in the spleno-myelogenous. In adults cases may be prolonged for many years, and some have ended in recovery. Hæmorrhages, diarrhœa, high fever, and general œdema are bad signs. Death often results from some intercurrent disease or from secondary infection. The spleen may diminish in size coincidently with improvement in the patient, or as death approaches. The differential diagnosis and the general treatment of anæmias will be considered in the next lecture.

Upper Brook Street, W.;
March 14th, 1904.

INTERNATIONAL TEXT-BOOK OF SURGERY. By WARREN and GOULD. (London and Philadelphia: W. B. Saunders & Co. Two Vols.)

The surgery of the English-speaking races seems to have fallen upon sad days. The exponent of surgery in these degenerate times is disinclined to grapple with the whole realm of surgery. He therefore advocates specialism as a necessity, and recommends some special operation which he has performed many times with slight mortality. The saddest effect of all this has been on our standard text-books. When we turn over the pages of Erichsen's great work, and find in it each subject treated with infinite pains and in a constant and good literary style, or when we take down from our shelves the text-books of Watson or Fagge, we find sound information conveyed in a uniform style and with a very high degree of literary excellence. The 'International Text-Book of Surgery,' and, indeed, all books of this type claim to be "a reliable text-book of surgery," produced by "men of wide experience and established reputations in the various departments of surgery," while they are in reality a compilation of articles on special subjects varying in excellence and in style. The articles on "Surgical Pathology of the Blood," "Fractures," and "The Surgery of the Spine" show care in their production. Dr. McBurney's article on "The Technique of Aseptic Surgery" is thoroughly practical. Rickets is a disease which always troubles the student, so indifferent is its description in nearly all text-books, and the account in this text-book is far from complete. The periosteal changes, although they account for the bending in the shaft of the bone, are dismissed in a few words in small print. The epiphysial changes are brought, as always, into undue prominence, and even these are diffusely described. Scurvy rickets is omitted altogether. In the article on "Diseases of Joints" the pathological changes in various joint diseases are not well explained: Charcot's joint is dismissed in a word or two, and there is no description of syphilitic joints. The article on cranial surgery is not very clear in many places, and in some passages the reader must be already well acquainted with the facts in order to grasp the meaning of the sentences. Many of the articles in the second volume are of a higher standard, but the work as a whole exhibits the shortcomings we have indicated.

WE have received from Messrs. Allen and Hanbury's some sample tins of the "Allenbury's" throat pastilles. In these pastilles the difficulty of meeting the demands of modern practice for a large variety of drugs in a soothing and demulcent form has been most efficiently solved. The form of the pastilles is rounded, and the composition is of the necessary softness, so that the preparations may be safely recommended in any case where an irritated or ulcerated mouth has to be treated. This desirable quality is obtained by the employment of a special "*pate de jujube*" which is soothing and palatable, and at the same time is of such consistency that a slow rate of solution of the pastille is ensured, so as to render more certain the local action of the drug incorporated in the mass. The pastille of chlorate of potash, borax, and cocaine affords a most useful medicament in relieving troublesome irritation and tickling of the throat, and undoubtedly presents itself to the mind as a particularly good combination for the kind of sore throat so often met with in public speakers and singers. In this pastille the stimulating action of the chlorate of potassium is admirably associated with the well-known characters of borax and the anodyne effect of cocaine. In the list of sixty-two pastilles medical men will find a well-arranged assortment of useful drugs.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 596. WEDNESDAY, MARCH 30, 1904. Vol. XXIII. No. 24.

CONTENTS.

PAGE

*Two Lectures on General Paralysis of the Insane. By GEORGE H. SAVAGE, M.D., F.R.C.P. LECTURE II 369

*Non-Tuberculous Apical Affections of the Lungs. By H. BATTY SHAW, M.D. 377

*Four Lectures on Normal and Abnormal Conditions of the Blood. By EDMUND CAUTLEY, M.D., F.R.C.P. LECTURE III—Intermediate Anæmias ... 380

** Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. Adlard & Son, Bartholomew Close, E.C. Telephone 927, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½ Bartholomew Close, London, E.C.*

TWO LECTURES ON GENERAL PARALYSIS OF THE INSANE.

Delivered at the Medical Graduates' College and Polyclinic,

By GEORGE HY. SAVAGE, M.D., F.R.C.P.,

Consulting Physician to Guy's Hospital; formerly Physician to Bethlem Royal Hospital.

LECTURE II.*

GENTLEMEN,—First of all as to the diagnosis of general paralysis of the insane. As I said before, it is practically the diagnosis between a certainly fatal disease and other conditions that may be curable. Therefore, in making the diagnosis it is of vast importance that you should be extremely careful, both from the social and from the medical points of view. Then, I would next say I intend to make distinctions between general paralysis as a whole and other forms of mental disorder. For instance, I will point out the difference between simple acute mania and the maniacal onset of general paralysis; simple curable melancholia and the melancholic phase of general paralysis; and dementia as a secondary disease—that is to say, secondary to other functional or ordinary forms of mental disorder,—and dementia due to the progressive decay of the brain. Now then let us look at the distinction between general paralysis and other forms of mental disease. Later I intend as far as I can to point out some of the more characteristic symptoms that occur in general paralysis, and to point out the difficulties that may arise in connection with them. Take as example one of the commonest symptoms described in general paralysis of the insane, so-called classical general paralysis of the insane, exaltation of ideas. I shall point out to you that there are

* Lecture given without notes, which were lost on the road.

Digitized by Google

other conditions under which exaltation of ideas may be met with. Pupillary inequality again is looked upon as a very characteristic symptom of general paralysis of the insane, but there are other mental diseases and disorders associated with inequality or irregularity of pupils.

To begin, I would say as to the stages of the disease, you will remember at the last lecture that I pointed out to you that there was what had been called a prodromal or threatening stage, a warning of general paralysis of the insane. A great deal has been made recently of so-called neurasthenia, a nerve exhaustion or nerve weakness; and within recent years it has been pointed out that one of the earliest symptoms of general paralysis is a form of neurasthenia. One has to remember that the brain is an organ that can express itself only in certain ways, therefore you may have, with a simple functional disturbance, the same symptoms as you have in organic disease. Take a simple example. Alcohol affects the same parts and the same tissues that are affected in early general paralysis of the insane. Therefore you get exactly similar symptoms resulting from alcoholism that you get from early general paralysis. The neurasthenia may be the result of temporary malnutrition, or more commonly of nerve-exhaustion from some special cause, or nerve-strain; or it may depend upon progressive loss of nerve-power. Therefore it is not at all surprising that an early stage of general paralysis of the insane may be associated with neurasthenia. Remember, then, if you have a middle-aged married man who has had syphilis years before brought to you with slight or comparatively slight functional nervous disorder, do not at once put it on one side and say, "Well, it is a temporary thing; it is a neurasthenia; it is a hysterical condition; it is a simple hypochondriacal condition, which will pass off." Do not, however, suppose that every male you see over forty is suffering from general paralysis; but at the same time be on the look-out, and do not be blinded by the fact that the symptoms are slight. How are you to distinguish between the onset of general paralysis and simple neurasthenia? In the same way that you would distinguish general paralysis from anything else. That there is not only a mental side but a physical side. If you have a man with slight mental symptoms or neurasthenia or hysteria and the like, and you get inequality of pupils, you get an alteration in the knee-jerks, more especially if you get irregularity of the knee-jerks and fixity of pupils, be on the look out. If, then, you find with the neurasthenia a distinct mental loss be suspicious; you know one of the characteristics of neurasthenia is that the individual, after a comparatively short rest, feels fit for anything. It is most astonishing to hear an individual after such an attack say, "I feel perfectly rested, and I feel fit to walk a mile." He walks a hundred yards and he is done. I have known leading medical men who have suffered from neurasthenia following perhaps influenza. After two or three weeks' rest the individual feels as fit as he has ever felt in his life, and his friends think he is as well. He comes back, and his intellectual abilities are as clear and as good as they were. But at the end of a very little strain at his work he breaks down. But still there is no intellectual defect. But the neurasthenic general paralytic would show a defect along his special lines. The artist, as I say, would no longer be clear in his drawing or exact in his colour; the singer would be faulty in one way or another; the artisan would show a defect in his most specialised power. Therefore remember that there is difficulty in the early stage of general paralysis very often in distinguishing between neurasthenia and simple general paralysis of the insane in the early stage of that disease. The same may be said of hysterical symptoms. I said in my last lecture that if you are called upon to see a middle-aged man who is markedly hysterical, be on the look-out. If, in the same way, you see a man otherwise healthy becoming emotional and hysterical and hypochondriacal, with a perfect certainty that he is never going to be any good to anybody again, or that some revolution has taken place in his inside, and if at the same time you find there is intellectual defect beyond passivity or stupor, and if you find alteration of knee-jerks or pupillary alteration, keep in your minds the likelihood of it being general paralysis of the insane. In speaking of these symptoms I am constantly laying stress upon the general conditions, and the chief one which you will say I am always repeating is the relationship to syphilis. I am obliged to do it, but I do not wish you to be misled into thinking that any very large proportion of persons who suffer from syphilis suffer from general paralysis of the insane. There

Digitized by Google

is all the difference in the world between the statement that general paralytics almost always have a specific history and the idea that many syphilitics suffer from general paralysis, for they do not. What the proportion is I do not know, but it is quite small; and, as I mentioned in my last lecture, there are whole tribes of Arabs and others who are as it were saturated with syphilis, and yet little or no general paralysis has been reported among them.

The next and probably the greatest difficulty arises with the maniacal onset. Here is a man or woman suffering from acute mania—sleepless, restless, excited, tending to irritability, tending to violence, destructive, amorous, and altogether maniacal. Is this simply mania, or is it mania as an early symptom of general paralysis? At first the mere mania, I think, has nothing characteristic as far as general paralysis is concerned. In nearly all cases, whether it be simply acute mania or whether it be mania associated with general paralysis, there has generally been a period of depression before it—the individual has been sleepless, restless, and wondering what was going to happen. He has been sure that some evil was impending—has been nervous in fact. And then comes, and perhaps suddenly, a reaction. He says, "What a fool I have been; I thought I was going to ruin; I thought everything was wrong. But now the whole is as right as possible." The general paralytic usually reacts more rapidly in this way. A maniacal patient may have been slightly depressed and may be slowly becoming restless, wilful, obstinate. And I would at once say that the characteristic, if there be a characteristic, of the maniacal general paralytic is that he is more easily led. The term used by Clouston in this connection is a good one, namely, "facility." You see the general paralytic dressed up in his wild extravagant garments, and with all his wildest ideas. You say to him, "Come along, I want you to go somewhere." He says, "Oh, but I cannot; I am going to do this and that and the other." You say, "Come along;" and he comes. Another patient, especially if alcoholic, will be inclined to say, "I am blessed if I go, for you or for anbody else; I am not going to be dictated to by you." So the general paralytic, with all his extravagant ideas, with all his exaltation, is generally more easily led. There are certain things in his mania that are very common; I would not say that they are characteristic, but they are very common. One of them is the tendency to write letters and send telegrams, and his marked amorousness. He is inclined to constantly and lightly make offers of marriage. He will write love letters by the half-dozen by the same post to actresses and singers, and sees nothing inconsistent in his conduct. The inconsequence of the acts of the general paralytic is very important. When we considered the melancholic cases of general paralysis we found there very often was an inconsistency. For instance, the individual who says he is ruined or says he has got obstruction of the bowels still eats and drinks and sleeps and gets fat. In the same way the general paralytic, with all his mania, is very inconsistent, more inconsistent than is the case usually with simple acute mania. Another symptom, and that I shall have to mention again later, refers to the exaltation and ideas of benevolence. The general paralytic not only has grand ideas, but he wishes everybody else to be equally happy and equally fortunate. If he has a thousand wives he tells you you may have five hundred of them. If he is well off in other respects he tells you you can have a commission on it. I mentioned to you last time the man who claimed to have the right to build the new Jerusalem. And another general paralytic quite benevolently said he had given him the job. I remember at Bethlem years ago an individual who believed himself to be archbishop of the world. At the morning service one day he stopped the chaplain and said he would make him Archbishop of Canterbury. He was quite benevolent with all his exaltation. The mania, therefore, of general paralysis is very acute. It is often associated with great restlessness, with great sleeplessness, with a tendency to write letters, a tendency to send telegrams, to amorousness, but also almost always a tendency to benevolence. The stage of acute mania with general paralysis is also marked by much tremor, particularly of the facial muscles. You will hear a great deal about tremor of the tongue and about small, fine, muscular tremors, but I do not think that that is at all characteristic. But the more excited the man is, the more his facial muscles work and the more his speech becomes affected. You get a great deal of tremor, of a kind which is different from the tremor which one meets with in alcoholic

Digitized by Google

cases. You see an alcoholic who is all tremor together, but you do not get that in the early maniacal stage of general paralysis. In these cases with mania as an early symptom of general paralysis of the insane you have to look for the inequality of pupils, alteration of knee-jerks, and alteration in the writing, and any defects of memory or of the senses which may be present. The maniacal stage of general paralysis not infrequently passes off more quickly than the acute stage of acute mania. The ordinary type of acute mania, especially if it has been preceded by some marked period of depression, lasts generally some months; whereas if I am called to see a case of acute mania with very great excitement, and I decide that the case is almost always certainly one of general paralysis, I warn the friends that this is almost certainly a fatal disease, but that the man very likely after a few weeks may be apparently so well that they may want to take him home. That sort of thing occurs; almost every week of my life I am seeing patients who are extremely maniacal, with all the exaltation of general paralysis, and so on, and it is so bad that they had to be sent off to the asylum on an urgency form. Within a few weeks the wife says, "You know my husband seems so very much better that I hope you will not mind seeing him again, because we think you may have made a mistake and that he is really well, and that you may now agree that there is no grave disease the matter with him." That constantly occurs. The more acute the maniacal symptoms are in early general paralysis the greater is the prospect, as a rule, of remission. So you have to be prepared for the disease if you see a case in which the actual maniacal symptoms rapidly pass off and leave the indivudal not quite himself but nearly himself. In such cases I make a fresh examination, to be sure I am not dealing with a case of general paralysis of the insane.

In the earliest prodromal threatening stage there may be tearfulness and depression, with the idea that something is going very wrong with the world, that there is something wrong with his digestion; and this slowly passes into the melancholic stage, with more or less hypochondriacal symptoms. Those are the cases, as I said before, which are more likely to mislead you than any others. The case is brought before one simply as one of melancholia. And I would repeat that if the patient is brought to you suffering from any form of mental disorder, if he belong to the group that is likely to produce general paralysis of the insane, be very sure that it is not general paralysis of the insane before you give an absolutely favourable prognosis. The two things in the melancholic form of general paralysis which you have to look out for are, first, the inconsistency between the symptoms and the result—the melancholic patient becoming fat and jolly in appearance, although he may go on repeating that there is something wrong with his inside. That was the case with the man whom I mentioned before, who believed that he was filled, that his bowels were stopped, and there was an enormous collection of fluid. Another patient in Bethlem used to pat himself on the abdomen with much satisfaction and shake his head and say he had thousands of tons of fæces in his abdomen, and yet he did not seem in the slightest degree concerned about it. There is therefore the want of consistency between the mental and the physical symptoms; the man complaining of being ill and in a poor way and yet all the time eating and drinking and sleeping well and getting fat. Again, you must be quite sure about the state of the pupils and the state of articulation.

The difficulty is greater in those cases where the melancholia is of the stuporous type. I freely admit that not infrequently I have been misled because I have not been able to examine the pupils or the reflexes. The individual will do nothing you want—you cannot get him to speak or write, and he is very like a log before you, an expressionless log, and you cannot tell whether there is any difficulty of control or any extra tremor about his tongue or lips, because he will not put his tongue out. It is clear, then, there may be very grave difficulties in such cases, and one would go a step further and say, if you have got a middle-aged melancholic with a syphilitic history who has not had previous attacks of mental disorder (and that I would say is a very important thing), it is possible he has general paralysis. As a rule the melancholic general paralytic is brought to the doctor with the history that he has never had mental disorder before the present attack. But if the case is one of ordinary melancholia between forty-four and fifty, there is often a history of previous attacks. You ask such a patient and he will probably tell you he has had two previous

Digitized by Google

attacks. The chances are, if the man has had two attacks of mental disorder during the last forty years or the last twenty-five years, he is not likely to be a general paralytic. Therefore remember it is all-important in examining cases of this kind, especially if it be the first attack and there is no obvious cause for it, such as alcoholism or plumbism, and especially if the melancholia be of the hypochondriacal type and if you get a history of syphilis in the case, be on the look-out for general paralysis of the insane. The same holds good exactly for cases of dementia. I intended to have enlarged more fully on these cases, and should have done so if I had not been unfortunate enough to lose my notes.

We have gone through the stages, now let us come to the consideration of some of the causes. General paralysis of the insane is associated more frequently with syphilis, therefore when you get a history of syphilis be on the look-out for this disease; but be on the look-out for other things. Remember that there are a good many other syphilitic neuroses or psychoses that may occur. The syphilitic neurosis or psychosis which we have to recognise in relation to general paralysis of the insane is progressive dementia. A certain number of cases who have had syphilis show evidence of either material degeneration or, what is less common now-a-days, gummatous affection of the brain or its membranes. In these cases you may have progressive dementia, and when you are asked to see a patient in a state of dementia it may be almost impossible for you at once to say whether it is the dementia of general paralysis or whether it is the dementia of progressive syphilitic brain disease. But it is all-important that you should know which it is from a prognostic point of view. Sometimes in the wards of Bethlem now I see patients who were admitted with syphilitic psychosis twenty-five years ago. I remember one such admitted in rather a silly hypochondriacal condition, with some loss of power on his right side, his speech became more affected, and although this recovered to some extent, and he regained almost completely the use of his legs and some slight amount of power in his arm, he has quietly passed into a quiet dementia, and lived for twenty years in that condition. He has had no further fits or further loss of power. If it had been a case of general paralysis depending upon syphilis, the course would have been a much shorter one. I shall have to postpone the fuller discussion of syphilitic psychoses and their symptoms to another lecture.

The most common cause of error is probably alcoholism. I told you just now that alcohol affects exactly the same parts of the brain that the poison, if it be a poison, producing general paralysis does, and therefore produces exactly similar symptoms. In fact, again to quote my old teacher, Wilks, who used to say, "When I am asked what are the symptoms of general paralysis of the insane, I say, 'Go to a students' dinner and watch the effects of alcohol on your friends.'" One becomes loquacious, loud and turbulent of tongue; another becomes pugnacious and aggressive; another becomes amorous and sentimentally poetical; another becomes emotional and tearful; another settles down to simple drunken stupidity. That is the course with alcohol and in general paralysis of the insane, and in some cases it is almost impossible to distinguish the two conditions. I saw the case yesterday of a barrister-at-law who was an alcoholic. He was a very hard worker, and had syphilis twenty years ago. He was chattering and mumbling away, and I felt that it might be the chronic maniacal condition of alcoholism. I do not know, and I do not think any one would be justified in saying yet, whether that man is suffering simply from alcoholism *plus* some degenerative change in the arteries, or if his disease is general paralysis started by alcoholism. So that you will have to remember, though I do not think general paralysis is started by alcohol pure and simple, yet I am sure it is common enough to have general paralysis on a syphilitic basis, and with alcohol as an exciting cause, just as it is not an uncommon thing, in my experience, to meet patients with a history of syphilis, alcoholic excess, and injury, who have an attack of influenza, which has been enough to start the degenerative process, and the end has been definite general paralysis. It is interesting to remember that lead affects the nervous system in almost exactly the same way that alcohol does. But though alcohol may affect the same part of the brain it is what may be called a transient poison: it passes off pretty quickly. Lead, on the other hand, is a more stable poison, and the result is that the symptoms, though they are sometimes like those of general paralysis produced by alcohol,

Digitized by Google

almost invariably pass off. They may leave a damaged cell, but they do not leave it damaged in the same way that the general paralytic poison does. Lead, in the same way, affects the same parts of the nervous system, and being a more stable poison the results are more lasting. Therefore in some cases you have true progressive general paralysis associated with fits, dementia, and the rest of the ordinary symptoms depending upon lead-poisoning. Among the more common difficulties are the distinctions between brain tumour and general paralysis of the insane. Some of the small multiple gummata affecting the membranes may implicate exactly the same parts of the brain that would be implicated under ordinary conditions by arterial degeneration and syphilitic infection, and you have precisely the same symptoms, and you could not tell the difference but for the fact that in some of these cases considerable amelioration takes place if you treat them anti-syphilitically, the symptoms being so exactly alike. As a rule you do not get the whole consensus of symptoms, and as a rule you get amelioration by treatment. And you also get more marked headache. In the diagnosis of general paralysis of the insane it is very important you should recognise the part played by headache. Headache, in my experience, is quite a rare symptom to be met with as an early or ordinary symptom of general paralysis of the insane. Though we say it is so frequently associated with syphilis, yet headache is an uncommon thing. And if there be headache it is not a nocturnal headache, it is not a severe headache, but rather a feeling of general confusion. These patients may say, "My head feels wrong," but acute or severe headache such as you get in syphilitic brain diseases and tumours of the brain are quite uncommon. As far as tumours are concerned, another important point of distinction is the optic-disc changes. As a matter of fact, I examine almost every patient I see with the ophthalmoscope, and have done so for years and years. Many years ago, with Dr. Rayner and Mr. H. Power, of St. Bartholomew's, I examined hundreds of cases at the county asylums, and I can only say it is the most uncommon occurrence to find optic-disc changes of any kind at all characteristic in general paralysis of the insane, unless it be quite in the last stages. In some of the cases where locomotor ataxy and ataxic symptoms have preceded mental symptoms for some considerable time, you may have blindness, you may find the optic-disc changes, but it is quite exceptional to do so. On the other hand, with brain tumour you know it is common to have such changes. Sickness is a common symptom with tumours of the brain, but it is quite an unusual thing in general paralysis of the insane. Again, with general paralysis of the insane it is quite unusual to get, when the disease is established, cranial-nerve implication. You know how common ptosis, and external strabismus, and mydriasis are in brain tumour cases, and in many of the syphilis brain cases; but in general paralysis it is most exceptional to find such symptoms. I think I told you in my last lecture that one of the earliest symptoms of general paralysis may be some temporary speech affection, some temporary affection of one of the cranial nerves, but that is some time before there has been any suspicion of general paralysis of the insane being present. It is all very well for me to lay down these laws so absolutely; but we learn more by our failures, probably, than we do by our successes. I think well and clearly of two patients in Bethlem Hospital at the same time. Both of them had malignant disease in some part of their bodies. In both cases there was a history of alcoholism; in both cases there was more than a suspicion of syphilis; in both there was some inequality of pupils, one probably due to old iritis, the other probably due to general paralysis. One could not get a view of the discs of either, and both of them were so far demented that it was impossible to get much out of them except purely objective symptoms. I diagnosed in the case of the first, general paralysis, and in the second, secondary cancer of the brain. The cases were such, but they were in the reverse order. The one which I had looked upon as general paralysis proved to be secondary growth of cancer, and that which I thought to be secondary growth of cancer was general paralysis of the insane. So under certain conditions I believe it is absolutely impossible, when you have a dement before you, to be quite sure whether you have to deal with a tumour, or whether you have general paralysis of the insane. In the same way, as I said last time in speaking about spinal forms of general paralysis, it is very difficult in the earliest stage of locomotor ataxy to be quite sure that you have

Digitized by Google

only got to deal with locomotor ataxy, and are not dealing with the locomotor-ataxy stage of general paralysis. In many cases it is almost impossible, from the symptoms, to decide whether the case is one of simple locomotor ataxy, and often you will have to say, "time alone can decide;" and time does decide. You have, in the majority of these locomotor-ataxic cases, a history of syphilis. You have probably contracted pupils, and you may have pupils which will not react to light; there are altered knee-jerks, and there may occasionally be maniacal outbreaks. I have seen several cases of maniacal crises occurring in locomotor-ataxic cases. So here you have everything to mislead you. You have a syphilitic history occurring in a middle-aged man who is married, with contracted non-reacting pupils, absent knee-jerks, and you have a maniacal outbreak. And yet that may, as I say, only be a mental crisis in a case of locomotor ataxy. Time and the nature of the delusions as a rule clear it up. There are locomotor-ataxic patients who become insane quite apart from general paralysis of the insane, and one has known them live twenty or twenty-five years. But in those cases, instead of having the general benevolent exaltation, they almost always have some type of delusion of persecution—they believe that their legs are converted by their enemies into lightning conductors; they believe that the devil is in some way pulling their legs off; they believe, if they have rectal crises, that some severe operation is being performed on their abdomen; if they have impotence, they believe that some vampire is sucking away their virility,—and such symptoms are not commonly to be met with in general paralysis of the insane.

Exaltation of ideas is looked upon as one of the most characteristic symptoms. I have told you that the exaltation has a more or less specific character. It is universal, it is generally benevolent. The man will ride a racehorse or preach in a cathedral; he will give you millions, or he will describe himself as being ten feet high and as being able to make you the same height if you wish. You have only to say what it is you want. He deals in large figures: millions of tons, a thousand wives, as many children as Solomon, and in fact the whole world is to be peopled by him, and they are to be the most beautiful and gracious race ever conceived. That is the general type of exaltation in general paralysis of the insane. You get exaltation in alcoholism to a certain extent, but it is of a more violent and aggressive type: "Get out of the way, I am stronger than you, I am not going to be interfered with by you." It is an aggressive not a benevolent exaltation as a rule. Then there comes the exaltation that occurs in what is called paranoia, delusional insanity, the nature of which is very interesting. A man may begin by being unduly sensitive and thinking people are all looking at him. He may be annoyed: "What on earth are they looking at me for? What is everybody noticing me for?" That is one stage. On the other hand, a person may become like a lady whom I saw this week, with whom her friends dared scarcely drive out in the streets of London, because she looks upon herself as being a princess, or at all events belonging to the Royal house, and bows to everybody. She has exactly the same feeling that everybody is looking at her, but instead of feeling it to be an offence she takes it as a compliment and believes herself to be a very important person. But it is a one idea. Years ago I remember such a one knocking my hat off because I did not recognise in him the king of Spain. He trotted about the area court like a small child with a stick, and was perfectly content with that one idea. If you said anything about his being king of England, he felt that was nothing to do with him; he was king of Spain. That slowly passed into monomania. And next I must notice another morbid growth of exaltation. A governess who, having been led to believe that mind is the measure of the man and of the woman, finds herself immeasurably above her employer; and then she begins to think, well, if right was right, I ought to be the important person, and these people ought to be lower people. Leading the solitary subjective life that so many governesses are forced to, slowly she develops the idea that she is what she wishes she were. So that one has seen a governess in Russia believe herself to be the Princess Bismarck. One has seen, under similar conditions, the wife of a bailiff to a nobleman, living in rather a small secluded cottage on the estate, believe herself to be the duchess. But there is no benevolence in those cases, there is simply single isolated exaltation. So the exaltation of general paralysis is as a rule general and benevolent. The exaltation of the ordinary type

Digitized by Google

of maniac is generally agressive. The exaltations of the person suffering from paranoia or from ideas of persecution and watching, are rather monomania than grandeur, without any benevolence and without any variability.

There are other things which have to be considered, and which I shall have to pass hastily over. I spoke to you about the difficulty of distinguishing between the symptoms seen in the alcoholic and those of general paralysis of the insane, and I would repeat to you that you have difficulties in all the stages. The early alcoholic looks extremely like a case of general paralysis. Chronic alcoholism may be very much in appearance like chronic general paralysis; you may get chronic nonsensical chatter, incoherent talk, and the chatter of the alcoholic may be very much like the chatter of the general paralytic. You may have a demented stage in chronic alcoholism, and that may be very much like that of the general paralytic.

The last symptom I shall have time to speak of to-day is the fits. Fits of all kinds occur at various stages of general paralysis of the insane, and I would impress upon you the importance of the occurrence of fits for the first time in middle-aged men. You may see cases, as I saw one this morning, of a girl whom I saw ten years ago, who had what looked like epileptic fits. Strontium and bromide seemed to have a very good effect for five years, but during the last few years she has had fits more frequently. That is the history of epilepsy, not an uncommon history. But if, on the other hand, you get a history of a man who has never had convulsions when an infant, who has not had fits, who has reached the age of forty-five or fifty without a fit, and then has a seizure of a distinctly epileptic type, then be on your guard, for I think the chances are then that it is either brain tumour or general paralysis of the insane. Suppose this man has a fit, is there any way of distinguishing between a fit due to general paralysis and one due to epilepsy? With regard to the fit itself, no! The same centre is affected and in the same way. But as a result, yes! The individual who is epileptic may have fit after fit, and it may be a long time before there is any intellectual loss. On the other hand, a general paralytic is almost invariably markedly worse in all his symptoms after a fit. If this man who had never before had a fit suddenly has one in his office and he is taken home, the next day he does not feel as well as the epileptic would—he complains that his tongue is sore, and he would not be ready for work for a day or two, and he would not feel as well as the epileptic. The fit would not pass off as clearly and as cleanly as would a similar visitation in an epileptic. Later on, when you have got to the maniacal stage, one has now and then to warn people that here is a man who is suffering from general paralysis of the insane, who is maniacal, violent, boastful, troublesome, and the whole of this may end in an epileptic or epileptiform fit. There again the fit will almost always lead to a change in the mental type. One great difficulty which you may have there is that with alcoholics you may get epileptiform fits as well; but the alcoholic more frequently recovers, and more completely than does the general paralytic. In the one case you have a greater structural change leading to the symptoms than you have in the other. I have told you that if you get hysterical attacks occurring in middle-aged men, be on your guard. Then comes the last form of fit which I would refer to, the cases of apoplectiform fit. And I must say that if you are called to see a patient suffering from an hemiplegic attack, I do not know how you are to distinguish and say at once whether it is the result of some hæmorrhage, some local softening, some tumour, or simple general paralysis of the insane. I think of an early experience of mine when I was called in to see a middle-aged woman who was in a fit. And they said she had had one before this, and it was thought she might have one more, based on the old faith that three fits was the limit which one person could endure. She seemed to be very bad. I was only twenty-three or twenty-four years of age then, and I was in country practice. I treated her *secundum artem*. She had mustard on her feet, turpentine enemas, and a few other things of the same kind, and I said I would see her next day. So I did, and found she was breaking everything. That was an apoplectiform seizure, associated in her case with alcohol and syphilis. You will therefore remember that whereas the fits of general paralysis, when epileptic in type, leave the individuals weaker than if they were simple epilepsy, on the other hand, the apoplectiform fits that attack an individual, making him hemiplegic, leave him much less damaged and more readily than if there had been a hæmorrhage. You can understand that—that it is an exaggerated epileptic fit with a one-sided local centre. Therefore you will find, if you are called to see a case of hemiplegia that passes off very rapidly with some mental excitement following, that you will have to be on the look-out for general paralysis of the insane.

Digitized by Google

NON-TUBERCULOUS APICAL AFFECTIONS OF THE LUNGS.

A Lecture delivered at the Hospital for Consumption and Diseases of the Chest, Brompton.

By H. BATTY SHAW, M.D.,
Assistant Physician to the Hospital and to University College Hospital.

GENTLEMEN,—It is freely admitted that for the successful treatment of pulmonary tuberculosis everything depends upon a diagnosis being made as soon as possible. With this object in view, all suspected cases are subjected to a careful physical examination, and it is practically certain that most physicians who in the course of such examination meet with hollowing of the upper part of the chest, with or without impairment to percussion, alteration of breath-sounds, and adventitious sounds, would have their suspicion aroused in favour of pulmonary tuberculosis. So important is the recognition of tubercle bacilli in the sputum that the next step would be to submit such sputum to microscopical investigation. Experience teaches that when symptoms point to a suspicion of tuberculosis of the lungs and physicial examination fails to detect positive signs or only equivocal ones, such as bronchial breathing at the right apex, a bacteriological examination is imperative. A case in point is a man, æt. 43, who will be shown to you presently; he is robust looking, well nourished, and well built. In June of last year he coughed up a "pint of blood," since then he has been troubled with slight cough and expectoration, usually "whitish," though occasionally slightly tinged with blood; repeated examination has failed to detect anything more than a few collapsed *râles* at the base of the chest, and an accentuated aortic second sound; the latter, however, has less than the usual significance in favour of consolidation of the right apex, because he has had syphilis and is by occupation a porter. To be on the safe side, his sputum was examined and found to contain tubercle bacilli; no doubt in his case, as in so many similar ones, the signs of pulmonary tuberculosis are masked by emphysema.

The cases to which your attention is specially called to-day are patients in whom there is no doubt as to the evidence of involvement of the apices of the lungs, because both signs and symptoms are such as to make the examiner very certain that there is some pathological change taking place. The sputa of these cases have been submitted to *bacteriological examination*, but in each case with negative results.

It must not be thought that these negative results are considered sufficient evidence of the non-tuberculous nature of the affection; for it is well known that there are fallacies, there may be tuberculosis of the lung and yet no true sputum derived from disintegration of lung tissue—the condition has not yet reached the stage of breaking down of the affected area, and the patient merely brings for examination the mucus derived from the upper air-passages; your patients will need instruction to prevent them presenting for examination the secretions of the naso-pharynx. Then, again, tubercle bacilli may be found at one time but not at another, so that in such cases it is imperative that several examinations should be made before a diagnosis is finally given.

Another matter of importance is that in certain cases a *tuberculin* test may be possible and desirable, but then of course, as most of you are aware, a positive reaction is much less valuable than a negative one, for the simple reason that a reaction may be given with tuberculin in patients who are free from tuberculosis, but suffering from malignant disease.

Again, the *family history* is important. A man, æt. 50, recently presented himself for slight hæmoptysis; he had had slight cough for many years, not sufficient, however, to need treatment or cause anxiety, but in the meantime it may be presumed that he was the cause of the decimation of almost the whole of his family, some having been carried off with meningitis or joint-disease, others with what was called consumption, and at the present time some members of his family are suffering from pulmonary tuberculosis. Such a case is not only a great argument for compulsory notification and rigid examination of the family, but it shows how a family history of tuberculosis in the children may be of help in calling attention to the serious condition of the parents.

A further test of the greatest importance is the *inoculation* of susceptible animals with the suspected sputum.

Digitized by Google

Most of the cases to be presently shown to you have been attending the hospital for years as out-patients (one for nineteen years); their sputa have always been free from tubercle bacilli; they have not lost flesh, or very little; their family history is good; in one case at least inoculation of the sputum into a susceptible animal gave negative results, and a similar negative result was obtained with tuberculin injection. Further, there is the unassailable indirect evidence that certain similar cases admitted to the hospital, and which have terminated fatally, have been found to be free from tuberculosis, although the apices of the lungs have been involved in disease. There is considerable reason, therefore, in holding the view that some of the present cases may be of the same non-tuberculous character. None of the cases shown to you are of an acute nature, for such cases are usually at once admitted to the wards; they are all chronic, just the sort of cases in fact that you are likely to meet with in routine practice, and in the urgency of other professional work you are liable to label as chronic pulmonary tuberculosis. Before demonstrating the present cases reference will be made to a number of cases, some acute, some chronic in course, which at first were considered to be tuberculous, but which ultimately were shown not to be so.

1. *Cases of apical croupous pneumonia.*—One of the first things you will learn in practice is that certain cases of acute consolidation of the lung associated with the presence of Fraenkel's diplococcus in the sputum are not severe in type, the patient even may be about and carrying on ordinary duties, though not feeling as well as usual. Four years ago a clerk came for advice; he had been ill for a fortnight, but was able to carry out his duties; latterly he found that he had lost weight and was sweating at night-time. On examination dulness was found at the right apex, with limitation of movement, bronchial breathing, and numerous consonating *râles*. The sputum was, however, free from tubercle bacilli and purulent. He was sent to the seaside, soon entirely recovered, and is now quite free from any pulmonary trouble. Another man, a carman, æt. 40, presented himself at University College Hospital for treatment for cough and hæmoptysis; the sputum was not merely rusty, it was markedly blood-stained, and, besides the ordinary signs of consolidation at the right apex, he even had a post-tussive suction sound over the same area. He was admitted to the hospital; no tubercle bacilli were ever found, though Fraenkel's diplococcus was readily demonstrated, and he subsequently left the hospital quite well, nor has he had any return of illness since.

2. *Malignant disease of the lung and mediastinum.*—Not many months ago a youth of very poor physique presented himself here, suffering from cough and expectorating scanty blood-stained sputum. There were evident signs of consolidation of the apex of the right lung; he brought his sputum at the next visit, but examination for tubercle bacilli was negative. Subsequently a small mass was discovered above the clavicle like an enlarged gland, and later on some contraction of the right pupil and right palpebral fissure was noticed. He was admitted to this hospital, and after some weeks died, and extensive lympho-sarcomatous disease was found in the mediastina, growing into the lung and involving the right sympathetic nerve. You will presently see a young woman, æt. 29, who came here in September, 1901, complaining of cough and dyspnœa of a month's duration; her father died of pulmonary tuberculosis. All that could be discovered was diminished movement of the right apex and a few scattered rhonchi in both sides of the chest. She was considered a suspicious case, but as there was no sputum with the cough, no examination could be made for tubercle bacilli. The patient did not continue her attendance here, and was lost sight of till lately. On February 6th of this year she again presented herself, complaining of weakness, slight cough, frothy expectoration, and very marked dyspnœa when lying on her back. As you will see, she has now general enlargement of the cervical and axillary glands, the veins of the front of the chest are enlarging, and there is obvious prominence and dulness to percussion over the upper half of the sternum. This case is probably one of lympho-sarcoma or lymphadenoma.

3. *Foreign bodies in the bronchus.*—About two years ago a man, æt. 45, came under my care at University College Hospital with violent cough and offensive expectoration. There was no history of having swallowed any foreign body. Air entrance was very deficient over the whole of the left chest, apex, and base, and at the base behind there were well-marked signs of what was considered to be excavation. Tubercle bacilli were found in the sputum. The patient died shortly after, and at the autopsy, as the left bronchus was being divided, a rabbit's vertebra was found impacted within its lumen; there was bronchiectasis at the base of the lung, and the whole lung was invaded with tuberculous disease. Possibly the foreign body had prepared the way for the tuberculosis as a secondary affection.

4. *Empyema.*—In the autumn of last year I was asked to see a patient in University College Hospital, who was exceedingly ill and presented a curious pulsating swelling at the front of the upper part of the left chest, the pulsation being synchronous with the heart-beats; subcutaneous emphysema was also noticed. On exploration with a needle very offensive pus had been removed, and a diagnosis of "empyema necessitatis" was made. At the operation a large amount of matter escaped, but neither then nor at the sub-

Digitized by Google

sequent post-mortem could any track be found in the chest-wall along which pus might have escaped from the interior of the chest, so that a more correct diagnosis would have been "pulsating empyema." Presumably infection had spread by lymphatics through the chest-wall and had led to diffuse subcutaneous suppuration, for pus was found lifting the whole of the superficial muscles of the back from the subjacent tissues. A "pulsating" empyema like this one was seen by Dr. Walshe, a former physician to this hospital, in 1843, and was described by him in his treatise on 'Diseases of the Lung and Heart,' and similar cases have been described by other writers.

There are other affections to which only a passing reference need be made, as there are no examples available at present to show you, but which, though of a non-tuberculous nature, present signs at the apices as well as at the bases of the lungs—aneurysm of the arch of the aorta may press on the left bronchus; pneumothorax may develop independently of pulmonary tuberculosis, and cases of gangrene or abscess are also met with. You will presently see an excellent specimen of a large emphysematous bulla occurring at the apex of a lung; such a condition would give rise to the so-called "pneumocele" occurring in children and adults the subjects of chronic cough.

5. *Cases of bronchiectasis.*—You will now be able to examine several cases in which signs of excavation at or near the apices of the lung form a well-marked feature, although tubercle bacilli are absent from the sputum. Judging from the amount of displacement of the heart and the retraction of the chest-wall, there is no doubt that they are combined with a considerable amount of fibrosis of the lung and thickening of the pleura. You will also be able to see specimens of bronchiectasis occurring throughout the lung apex and base, some uncomplicated and others combined with tuberculosis; further, two specimens will be shown which will dispel any doubts you may have as to the occurrence of apical bronchiectasis; the direct continuity of the cavities with the bronchi is obvious, and Dr. Shrubsall tells me that microscopic examination of the tissues in the neighbourhood has failed to demonstrate any tubercle bacilli or tuberculous granulomata. One of the patients shown to you to-day, a tie-maker, has been attending this hospital for nineteen years for cough, etc.; you will see by Dr. Habershon's notes on the out-patient letters that she has had apical signs for at least nine years. The patient is æt. 42, and weighed a year ago as much as she did five years previously, though she has lost weight a little lately. Symptoms referable to the lungs are not marked, and she last sought advice for dyspeptic symptoms; tubercle bacilli have not been demonstrated. Another patient, a married woman, æt. 35, has had signs over the position of the middle lobe of the right lung for the last seven years; there are signs of a cavity in this area, and the rest of the side of the chest is impaired to percussion. The sputum has been examined eight times, but always with a negative result. There is no history of tuberculosis; since attending this hospital she has married and given birth to a healthy child. The next patient, a woman æt. 42, shows dulness over the whole of the left chest, with obvious excavation at the left apex and dislocation of the heart to the left. Tubercle bacilli have never been demonstrated, either in the hospital or as an out-patient, and I failed to get positive results on inoculating a guinea-pig with the sputum in August of last year. The two male patients show very similar physical signs, with the same absence of tubercle bacilli in the sputum, and in the younger one you will find that the heart is dislocated entirely to the right of the sternum. Both men have been ill for five years, and the family history in each case is quite negative as far as tuberculosis is concerned. The elder man is a stonemason, and it is conceivable that his lung condition is one of pneumoconiosis. It is usual to find that cases of bronchiectasis complain of copious expectoration of a very offensive nature; the absence of this symptom in these cases may be due to the more effective drainage of the dilated tubes when present at the apices of the lungs. I do not attempt to discuss the question whether possibly these patients are cases of healed apical tuberculosis or not; at the present moment they are non-tuberculous, and the history and clinical course of these cases justify one in calling them non-tuberculous apical affections of the lung. The suggestion is made that they are bronchiectatic, but to establish this completely post-mortem evidence would be required.

One final word of warning. It is your duty to call all such cases tuberculous until a thorough examination of the sputum and application of the tuberculin test justify you in holding a brighter view of the case. I should be guilty of the charge of disseminating wrong principles if I left you with the impression that you might diagnose these cases at once. There is little or no difference between the signs met with in these cases and those of the ordinary fibrotic form of chronic pulmonary tuberculosis. Their great characteristic is extreme chronicity and absence of tubercle bacilli in the sputum. If such cases do exist, and I hope you have been convinced by the pathological and clinical demonstration of their existence, then you may rest satisfied with the idea that you may be able to rescue from the limbo of protective social ostracism certain cases which, though seriously hampered by their apical disease, need not be segregated and exposed to the trials of special treatment against tuberculosis.

Digitized by Google

FOUR LECTURES
ON
NORMAL AND ABNORMAL CONDITIONS OF THE BLOOD.*

By EDMUND CAUTLEY, M.D.Cantab., F.R.C.P.Lond.,
Physician to the Belgrave Hospital for Children and the Metropolitan Hospital.

LECTURE III.
INTERMEDIATE ANÆMIAS.

GENTLEMEN,—Under the heading of intermediate anæmias are included various forms of anæmia of uncertain origin. Some are with difficulty separable from the secondary anæmias, while others in their course and characteristics are more closely allied to the primary anæmias. Their nomenclature is most confusing, different names often having been given to conditions which are apparently identical, and the same name to affections which are quite dissimilar. Thus we find cases described under the name of splenic anæmia (Griesinger, 1886), anæmia splenica infantum, anæmia splenica infettiva dei bambini, pseudo-leukæmia splenica, anæmia infantum pseudo-leukæmica or von Jaksch's disease; Hodgkin's disease, lymphoma, lymphadenoma, lympho-sarcoma; splenic lymphadenoma, l'epithéliome primitif de la rate (Gaucher, 1882), s éno-mégalie primitive (Bruhl and Debove, 1892); lymphatic anæmia. It is difficult to differentiate strictly between the different varieties, but they can be fairly clearly described under three headings. A fourth group of cases is also given, though it is not generally accepted as a special variety.

I. ANÆMIA SPLENICA INFANTUM.—Under this name I include cases of anæmia, associated with enlargement of the spleen, without any definite evidence of a primary cause and without any characteristic changes in the blood beyond a certain polymorphous character. The cases are most common in infants between the ages of six months and two years. Of the last seven which have come under my notice the youngest was ten months and the oldest twenty-two months. Four of them were males and three of them were females.

Symptoms.—The onset and course are like those of other forms of anæmia. It starts and progresses insidiously, the pallor being the most characteristic feature. In consequence of the anæmia the patient acquires a waxy complexion with a tinge of olive-green in it, dyspnœa, dilatation of the heart, and hæmic murmurs. The child may remain plump though the anæmia and debility steadily increase. Some are very emaciated. The abdomen is protuberant and tympanitic, the enlargement being mainly due to the enlargement of the spleen, which may extend right down to the left iliac fossa. The splenic hyperplasia may be the first indication, arising even before the anæmia. Occasionally a bruit is heard over it. Perisplenitis may also occur, and attacks of paroxysmal colicky pain in the left hypochondrium, due to traction on the abdominal sympathetic plexus or to twisting of the peritoneal attachments. The liver is usually enlarged and extends from one to two inches below the costal margin. Sometimes the lymph-glands are a little enlarged. Hæmorrhages are not frequent, though epistaxis and subcutaneous hæmorrhages are liable to occur during the course of the disease, and gastro-intestinal hæmorrhage in late stages. Pyrexia is variable, irregular, and usually due to complications. Gastro-intestinal symptoms are common, such as impairment of appetite, vomiting, and the passage of undigested food. Jaundice may result from catarrh of the duodenum. The urine is normal or contains a trace of albumen. Œdema of the feet occurs in late stages.

Ætiology.—Whether these cases are merely forms of secondary anæmia or are due to some independent poison, a toxin, which gives rise at the same time to anæmia and splenic enlargement, is at present uncertain. The disease is frequently associated with rickets to a variable degree. Thus, in the seven cases referred to, rickets was present to a slight extent in all of them and was well marked in two. Riviere ('Med.-Chir. Trans.,' vol. lxxxvii, 1903) found it present and well marked in six cases out of thirteen and only absent in one. On the other hand, it must be borne in mind that rickets is a very common disease, and even in its most marked forms is usually unassociated with splenic enlargement. Also it must be remembered that the same conditions which give rise to splenic anæmia may assist in the production of rickets. Some cases have been ascribed to congenital

* Delivered at the Medical Graduates' College and Polyclinic.

Digitized by Google

syphilis. Thus Monti and Berggrun found a history of syphilis in sixteen out of twenty cases. In my last seven cases there was neither a family history nor any direct evidence of syphilis. It is a curious fact that the disease is more common among Jews, especially Polish Jews, than among Gentiles. It is also most common in hand-fed children, especially those brought up on proprietary foods and condensed milks. Often there is an antecedent history of digestive disturbance. It is quite possible that unsatisfactory methods of feeding and bad hygienic surroundings are sufficient to account for the condition, and that we are simply dealing with a variety of secondary anæmia rather than with a special entity due to the action of an unknown intestinal toxin.

Examination of the blood.—The blood shows no pathognomonic change beyond that of secondary anæmia of the chlorotic type and polymorphism. Thus, the number of red cells is usually reduced to below 3,000,000, and may fall as low as 800,000. The cells vary much in size; there are many microcytes, megalocytes, poikilocytes, and usually polychromatophilia. The hæmoglobin may fall to 30 per cent. or even lower; the colour index is low; hæmatoblasts are always present, chiefly in the form of megaloblasts and normoblasts. Karyokinesis is common, and is regarded by some as a diagnostic feature. The number of leucocytes varies. There may be an actual leucocytosis up to 50,000, or a relative one in which the proportion reaches 1 to 100 red cells, or even 1 to 12. Sometimes the number of leucocytes is actually deficient. The white cells exhibit great variety in size, shape, and staining properties. The variation is more marked in the polynuclear cells than in the lymphocytes. Eosinophiles are deficient (von Jaksch), normal or increased (Riviere), increased (Luzet). "Mast-cells" are often found, and myelocytes may be present in advanced stages, even in large numbers. Thus it is evident that a diagnosis cannot definitely be made from the blood examination alone. The presence of myelocytes and hæmatoblasts is not diagnostic. We might, however, be justified in limiting the name to those cases in which there is a definite leucopenia, while grouping all those in which leucocytosis is present under the heading of secondary anæmia. The great objection to isolating such a group arises from the fact that a leucocytosis may appear in a typical case as a result of fever or of a secondary infection. Riviere is inclined to lay great stress on the polymorphous character of the blood, polychromatophilia and great poikilocytosis. In its polymorphous character it differs from the blood of secondary anæmia.

Morbid anatomy.—The spleen is large, usually enormous, and has a firm rounded edge. Under the microscope it shows simple hyperplasia without any leukæmic infiltration. The liver is generally larger than normal, but is not enlarged in proportion to the size of the spleen. Microscopically it resembles the fœtal liver. The lymph-glands show a little hyperplasia in about half the cases. No characteristic changes are found in the marrow. There is no post mortem evidence in any organ of true leukæmia.

Under the title of *Anæmia Infantum Pseudo-leukæmica* Von Jaksch ('Wien. klin. Woch.,' 1889, Nos. 22, 23) attempted to isolate a special form of anæmia in infants. According to his description, this disease is characterised by anæmia, great splenic hyperplasia, a slight or moderate increase in the size of the liver, some glandular enlargement in about half the cases, and marked changes in the blood. The percentage of hæmoglobin is deficient, the number of red cells is diminished, and leucocytosis is marked. The symptoms and course are the same as those of true leukæmia, except that it is fatal in only about 20 per cent. of the cases. No leukæmic changes are found post mortem. He regarded it as a primary form of anæmia, and laid stress on the relatively slight increase in the size of the liver, compared with the spleen, as an important diagnostic feature in which it differed from true leukæmia. As a matter of fact, this is not of any diagnostic value, for the liver in true leukæmia varies very much in size.

Since then many instances of the disease have been recorded, but it is by no means clear that they are identical with the affection as originally described. The existence of the disease as a special entity remains doubtful. Many observers believe that such cases are merely secondary to many varieties of anæmia with splenic enlargement. There is no doubt that many varieties of anæmia, usually secondary, in which the spleen is enlarged have been described under this name. In other words, some of the cases are merely severe forms of secondary anæmia with leucocytosis. Ætiologically cases are found just as frequently associated

Digitized by Google

with rickets as those described under the name of anæmia splenica infantum. Fischl ('Zeit. f. Heilkunde,' 1892) found inherited syphilis in two and rickets in sixteen out of eighteen recorded cases. According to von Jaksch the disease is most common between six and twelve months of age, and occurs up to the age of four years. It is more common in girls than in boys. On the whole we had better regard von Jaksch's disease as merely another name for the disease which I have described as anæmia splenica infantum.

Diagnosis.—A pure case of anæmia splenica infantum should give no history or evidence of rickets or syphilis. No doubt many of the cases are simply severe forms of secondary anæmia, but there may be cases that cannot be classified under any other title. Some may appear to be intermediate between primary and secondary anæmia, while others may at times exhibit the characteristics of a true leukæmia. Changes in the red cells are not characteristic, and may occur in any form of grave anæmia. Luzet regards a large number of hæmatoblasts and the presence of karyokinesis as important diagnostic signs. The leucocytosis is not characteristic; it is merely one of degree, and is not greater than is found in many secondary anæmias. From leukæmia it is differentiated by the relative count of the white cells. The presence of a large number of myelocytes may lead to an erroneous diagnosis of leukæmia. The relative degree of increase in the size of the liver and spleen is of no value, for it may be no greater than is seen in some forms of true leukæmia.

The *prognosis* is good, though uncertain, as the cases generally occur in the poorer classes and are lost sight of. Hence we have not sufficient data as to their subsequent progress. Cases are chronic and tend to relapse. The presence of hæmorrhages and the occurrence of œdema are bad signs. I generally regard minute subcutaneous petechial hæmorrhages on the abdomen and trunk as a very bad, though not necessarily fatal indication. Usually death occurs from some intercurrent disease, such as bronchitis, broncho-pneumonia, or general tuberculosis, or from simple exhaustion. Gastro-intestinal derangement is frequently conducive to a fatal issue. If the leucocytosis is moderate and there is no complication, the child will probably recover. A progressive increase in the leucocytosis may occur in fatal cases, or leucopenia may be present throughout. In a boy, æt. 13 months, under my care, a large number of myelocytes were found on one occasion, and the child was extremely ill, but in spite of the profound blood-changes he eventually recovered. In one case, in which I had the abdomen opened with a view to removal of the spleen, it was found that there was in addition miliary tuberculosis of the peritoneum. Of my last seven cases, only one died in hospital, but what has become of the others since I do not know. The spleen may contract to its normal size.

II. Hodgkin's Disease.—This affection is characterised by progressive hyperplasia of the lymphatic glands and anæmia. Usually the liver and spleen are enlarged, and contain lymphoid masses resembling secondary growths. Similar masses may be found in other organs. In its mildest type it may be limited to one group of glands, more especially the glands in the neck, and is then frequently named *lymphoma*. When generalised it resembles true lymphatic leukæmia clinically, but the blood-picture is never that of a leukæmia. At first the blood is practically normal. Later it exhibits the usual characteristics of secondary anæmia. The number of red cells may fall to two million, the colour index is low, and in advanced cases a few normoblasts are formed. The white cells are normal or diminished in number, but in some cases, especially towards the end, a simple general leucocytosis may be present, or an inflammatory leucocytosis—that is, a relative increase in the polynuclear cells,—as a result of a secondary infection. Cases may be acute or chronic. In the former the ordinary symptoms of the chronic form are associated with moderate pyrexia and a hæmorrhagic tendency.

The disease is frequently diagnosed in infancy, but it is probable that many other conditions of lymphatic hyperplasia are wrongly included. Many supposed cases are really tuberculous, while some may be syphilitic, or even malignant, such as lymphadenoma and sarcoma. The acute cases suggest the possibility of an infectious origin, and in some of the chronic cases local sources of infection have been found in the mouth, middle ear, etc. Thus, in the case I referred to in my last lecture, the disease apparently started as an infection in the middle ear, and later on there was secondary infection by the tubercle bacillus, and

Digitized by Google

possibly even by septic micro-organisms. The diagnosis must be based upon the presence of more or less general glandular enlargement, moderate enlargement of the spleen and liver, anæmia, and the characteristics of the blood examination. The latter are of negative rather than of positive value by enabling us to exclude the various forms of leukæmia in a case which is clinically one of true leukæmia. It must not be forgotten that a few myelocytes may be present in advanced cases. The glands most often affected are those in the anterior and posterior triangles of the neck. In addition we frequently find the glands in the axillæ and groin enlarged, and those in the mediastinum and mesentery. The glands are firm, movable, not adherent to the skin or to each other, and do not tend to break down except as a result of secondary infection. On the other hand, glands enlarged from tuberculous infection adhere to the skin and to each other, and are liable to caseous degeneration.

The prognosis is bad, except in the mild cases limited to one group of glands. Acute leukæmia has been known to occur in the course of the disease (vide Lecture II), but is rare. Leucocytosis is liable to occur from secondary infection, and may give rise to a mistaken diagnosis of leukæmia—a disease which should not be diagnosed unless a blood examination shows that the leucocytosis is typical of true leukæmia. If not, the leucocytosis must be regarded as inflammatory or as the leucocytosis of the dying.

Lymphadenoma.—Although this is often included under the name of Hodgkin's disease, it may differ from it, and may even simulate myelogenous leukæmia. Or the leukæmia may develop as a secondary process. Rolleston and Latham recorded ('Lancet,' 1898, i, 1313) a case in a boy of eighteen months, in which the stomach was involved. Apparently it was secondary to middle-ear disease, was complicated by rickets, and closely simulated myelogenous leukæmia. The blood showed poikilocytosis, many normoblasts, and a few megaloblasts; much leucocytosis, with a blood-count of lymphocytes, 61·1; polynuclear cells, 16·5; eosinophiles, 1·6; myelocytes, 20·8. Post mortem the stomach was found covered with numerous smooth, white, polypoid growths all over the lining membrane except near the pylorus. The growths varied in size, up to that of a walnut, and were composed of mucous membrane thrown into folds and extensively infiltrated with large and small lymphocytes, apparently due to proliferation of the lymphoid tissue of the mucosa. A mass of enlarged glands was present under the sterno-mastoid. The spleen weighed eight ounces. The costo-chondral cartilages showed excess of lymphocytic infiltration. In this case the morbid anatomy indicated lymphadenoma, while the blood examination indicated myelogenous leukæmia. The large percentage of myelocytes is said to be only found in true leukæmia, though I have come across a similar high percentage in a case of anæmia splenica infantum which ended in recovery.

III. Anæmia Splenica of Banti.—Banti described three cases in 1881 under the name of progressive anæmia or anæmia ganglionare. Several other cases have been described since. They are rather more frequent in males than females, and usually occur in adults. Of thirty-five cases the average age was thirty-two years. Only two of these were children. It may occur in more than one member of a family, affecting several children, but not necessarily beginning until adult life. Six in a family of three generations had large spleens (Claud Wilson); two sisters (Boivard); two in family (Collier). Cases are often described under the name of splenomegaly. Syphilis, malaria, and alcoholism do not appear to have any direct influence, but may act as predisposing causes. The characteristic symptoms are anæmia and enlargement of the spleen, with secondary cirrhosis of the liver and ascites. Epistaxis, bleeding from the gums, hæmatemesis, and melæna are likely to occur. The hæmorrhages are characteristic and repeated at intervals. They are usually nasal or gastro-intestinal, hæmatemesis and melæna being the most common and the most severe. In a case of Osler's they recurred for twelve years. Various theories have been put forward as to the causation. Thus, Banti ascribes it to lymphoid infiltration of the ganglia of the neck and semilunar ganglia, and the solar plexus in the abdomen. Senator ('Berl. klin. Woch.,' 1901, xlvi) regards it as an ordinary splenic anæmia with cirrhosis. Barr ('Lancet,' 1902, ii, 496) ascribes it to a vaso-motor paresis of the splanchnic area due to disease of the visceral sympathetic ganglia. Rolleston ('Clinical Journal,' 1902, xix, p. 401), on the other hand, regards the disease as a special entity, and restricts the name to cases due to endothelial proliferation in the spleen. He recorded a case in a man of twenty-three who had been in a children's hospital twelve years before with much the same condition. The spleen weighed thirty-seven ounces, and microscopically the normal tissue was found to be largely replaced by fibrous tissue and numbers of large endothelial cells with two or more nuclei, the endothelial cells being due to proliferation of the cells lining the trabeculæ and the walls of blood-sinuses. The condition has been described as primary carcinoma of the spleen by Gaucher in 1882 and Picon and Ramond in 1896. Possibly it is due to a toxic or infective cause.

IV. Lymphatic Anæmia.—A form of anæmia has been described by Alex MacGregor ('Lancet,' 1901, i, 921) as a morbid entity under the name of lymphatic anæmia. Of twenty-three cases, between

Digitized by Google

the ages of three and sixteen, eighteen were under eleven years. It is much more common in boys. It is characterised by marked adenitis, which is often overlooked, for the glands are small and hard. They are usually found in the cervical triangles and the groin. The spleen is usually enlarged. The disease is insidious, chronic, and ends in recovery. The blood is deficient in red cells and hæmoglobin. The white cells are increased, especially the large lymphocytes. The count shows small lymphocytes, 12 to 13, large 20 to 40; polynuclear, 41 to 63; eosinophiles, 2 to 7. Thus in these cases we have a lymphocytosis of large cells.

The symptoms are those of anæmia, with clean tongue, poor appetite, constipation, loss of flesh, and cough which is worse at night. Possibly the cough is due to enlarged bronchial glands. Probably these cases were simply mild forms of secondary anæmia. They all recovered, and none developed tuberculosis.

General Diagnosis.—In the first place certain diseases must be excluded when we have to deal with anæmia. In malaria we find no leucocytosis, and there are definite clinical symptoms and a parasite in the blood. In rickets we find anæmia of the chlorotic type. The blood-picture may resemble that of anæmia splenica infantum. Occasionally we meet with leucocytosis up to 40,000 without any anæmia. Hence, in the presence of rickets it is impossible to be certain whether the blood-state is due to the rickets or to the anæmia. In congenital syphilis we frequently find anæmia with considerable reduction in the percentage of hæmoglobin and the number of red cells. Hæmatoblasts are often present, and may be abundant. Leucocytosis and lymphocytosis may be very marked, and, if the skin lesions are severe, there may be eosinophilia. In tuberculosis there is a simple deficiency in red cells and hæmoglobin, with a leucocytosis if any secondary infection occurs.

Next we must consider the positive value of the blood examination in diagnosis. The occurrence of hæmatoblasts is not conclusive of any one particular variety, for they are common in all forms of severe anæmia, and in the worst cases are associated with degenerative changes and polychromatophilia. Myelocytes in large numbers usually indicate spleno-myelogenous leukæmia, but they may also occur in advanced cases of secondary anæmia and anæmia splenica infantum in sufficient numbers to suggest true leukæmia. They appear much more readily in children than they do in adults. If, indeed, they are so excessive as to reach 30 to 50 per cent. of all leucocytes, the case is undoubtedly one of spleno-myelogenous leukæmia. But it must not be forgotten how rare this latter disease is in infancy. Very marked lymphocytosis indicates lymphatic leukæmia. Pernicious anæmia can be diagnosed by its progressive course in conjunction with severe progressive oligocythæmia, oligochromæmia, and the presence of myelocytes, with little or no splenic enlargement.

In Hodgkin's disease the glandular enlargement is almost invariably marked, and there are no characteristic blood-changes. So, too, in lymphadenoma.

Banti's disease is too rare to require further notice. It may be mistaken for cirrhosis of the liver.

We are hardly justified in regarding all cases of anæmia in infants as of the same type and merely differing in degree. Such a view has been put forward by Riviere, with the suggestion that the ætiology consists in the absorption of intestinal toxins. That one form of anæmia may pass into another is quite possible; so, too, may general tuberculosis follow a capillary bronchitis. But there are sufficient differences to warrant a classification such as given above. Until we know more about the exact ætiological factors concerned in the production of the various forms of anæmia and splenic enlargement it would be rash to group all cases under one name. Probably some of the varieties depend upon the coincident or consecutive action of more than one ætiological factor.

Treatment. — The treatment of all cases of anæmia consists in putting the patient under the best hygienic conditions possible and on a suitable diet. If a definite cause can be diagnosed it must be carefully attended to. Do not treat the symptoms and neglect the disease. In bad cases, or even those of moderate severity, complete rest in bed is essential. Moderate massage can be used to replace exercise. The two drugs necessary are iron and arsenic. The iron may be given in the form of inorganic drugs, *e. g.* saccharated carbonate and tartrate of iron, in various proprietary mixtures made from blood, or in the form of organic compounds in food. Of these the latter are the most readily assimilated, and are the most valuable. Thus I should advise you to give yolk of egg, bone-marrow, raw meat or meat-juice, and potatoes, all of these being foods which contain a considerable quantity of iron. Arsenic may be given, and appears especially suitable in cases of pernicious anæmia, and, to a less extent, in leukæmia.

Operative treatment.—This is rarely justifiable except in cases of Banti's disease. Removal of the spleen in cases of this nature may be followed by recovery, whereas if it be removed in true leukæmia the patient soon dies, and in the cases of anæmia splenica infantum such an operation is unnecessary and useless.

Great care must be used to avoid chills and to guard against cardiac syncope in bad cases. Cod-liver oil, Syr. Fer. Iodidi, Syr. Fer. Phosp. Co, and maltine are all useful in cases of secondary anæmia and anæmia splenica infantum.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 597. WEDNESDAY, APRIL 6, 1904. Vol. XXIII. No. 25.

CONTENTS.

PAGE

*On the Differential Diagnosis of Chronic Disorders of the Stomach. By GEORGE HERSCHELL, M.D. 385

*With Dr. Campbell Thomson at Middlesex Hospital. A CASE OF INJURY TO THE CEREBRAL CORTEX ... 392

*Four Lectures on Normal and Abnormal Conditions of the Blood. By EDMUND CAUTLEY, M.D., F.R.C.P. LECTURE IV.—The Thymus Gland ... 395

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone 927, Holborn.

All inquiries respecting Advertisements should be sent to Messrs. Adlard & Son, Bartholomew Close, E.C. Telephone 927, Holborn.

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15s. 6d. per annum; Abroad, 17s. 6d.

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Ltd."*

Reading Cases to hold Twenty-six Numbers of THE CLINICAL JOURNAL *can be supplied at 2s. 3d. each, or will be forwarded post free on receipt of 2s. 6d.; and also Cases for Binding Volumes at 1s. each, or post free on receipt of 1s. 3d., from the Publishers, 22½, Bartholomew Close, London, E.C.*

ON THE DIFFERENTIAL DIAGNOSIS OF CHRONIC DISORDERS OF THE STOMACH.*

By GEORGE HERSCHELL, M.D.Lond.

GENTLEMEN,—I feel sure that I need offer no apology for selecting as my subject the differential diagnosis of disorders of digestion, as these affections form so important a part of our daily work. As it would be presumption on my part to attempt to treat exhaustively a subject of such magnitude in the time at my disposal, I shall confine myself to outlining in a general way a practical method for everyday use of arriving at as definite conclusions as the circumstances will justify in the cases which present themselves for treatment.

We cannot too persistently keep the fact before our minds that we have no right in the present state of knowledge to group all the functional disorders of the stomach under the heading "dyspepsia." In the first place, it simply begs the question, as such a diagnosis is merely a statement in other words of the fact, well known both to ourselves and the patient, that the digestive process is not carried on as it should be; and, secondly, we are not justified in remaining satisfied with such an imperfect apology for a diagnosis when the resources of modern medicine will enable us to acquire absolute data upon which to base our opinion. In every case we can, at least, ascertain exactly what happens to the food after it enters the stomach, and the length of time which elapses before it enters the duodenum, and the knowledge thus acquired should help us materially not only in making our diagnosis, but in inaugurating a rational scheme of treatment.

* An address delivered before the Willesden and District Medical Society.

Digitized by Google

One of the most striking differences between the medical practice of ourselves and that of our grandfathers lies in the fact that whilst they attempted, with more or less success, to distinguish one disease from another by a laborious analysis of subjective symptoms, our opinion is professedly based upon logical deductions from definite data obtained by modern methods of examination. This is especially the case in diseases of the stomach, but even here it must not be supposed that we must use intra-gastric methods indiscriminately as a routine practice in all the cases of deranged digestion which come under our notice. It will frequently happen that these are neither advisable nor necessary, and, apart from cases where the use of the tube is contra-indicated, when we can diagnose a case of slight severity and short duration with reasonable certainty by the inductive mental processes, we should certainly do so, and not subject our patients to the annoyance of instrumental interference.

For practical purposes we may thus divide our cases into two distinct groups: those which do, and those which do not, necessitate the use of intra-gastric methods for their elucidation.

(A) Cases not Requiring Intra-gastric Methods.

(*a*) *Slight Temporary Derangements of Digestion obviously due to Errors of Diet or some other easily ascertained cause.*

Our diagnosis will be confirmed by the fact that these affections prove transient, and promptly recover under appropriate treatment. We must, nevertheless, always keep before us the possibility of such illnesses being in reality the characteristic attacks of gastric insufficiency which mark the earlier stage of pyloric stenosis or gastric myasthenia. The regularity with which they recur, and the invariable manner in which they follow excess in diet will point to the necessity for a complete examination.

(*b*) *The Slighter Forms of Gastric Neurasthenia or Nervous Indigestion.*

The different effect at different times of the same articles of diet, the evident dependence of the symptoms upon the nerve tone of the moment, together with the characteristic symptom-complex of neurasthenia, will usually lead to a correct diagnosis. In such a case the administration of a test meal with the subsequent examination of the stomach contents would not aid us very materially in forming our diagnosis, as the composition of the gastric juice is well known to vary considerably at different times even in the same case. The slight myasthenia so frequently present can be recognised by the use of a simple effervescing powder.

(*c*) *Cases of Simple Hyperchlorhydria in Young Men.*

The symptoms of this condition are so well marked that mistake is almost impossible. The epigastric pain coming on one or two hours after food, relieved by alkalies and albuminous food, its termination in many cases by the vomiting of an acid liquid, the limitation of pain to the digestive period, the absence of painful pressure spots or cutaneous hyperæsthesia, and the absence of blood in the vomit or stools all point unmistakably to this affection. Probably there will also be a history of the excessive use of spirits or tobacco.

(*d*) *Dyspepsia in Chlorotic Girls.*

In these cases the trouble usually consists of hyperæsthesia of the mucous membrane of the stomach with or without hyperchlorhydria. If the pain follow immediately after the ingestion of food and does not undergo any exacerbation later on, we have probably hyperæsthesia only; if it is increased a couple of hours after food, when the hydrochloric acid becomes free in the stomach, there is most probably hyperchlorhydria. This distinction, unfortunately, is not absolute, as in many cases of hyperæsthesia a normal amount of free acid will give rise to pain. But there is really no necessity to ascertain whether there is an excess of hydrochloric acid, as in all cases where the pain increases at the height of digestion the proper treatment is the administration of alkalies. In all these cases the chief and most important problem is to ascertain whether there is ulceration. This subject will be discussed in the following paragraph.

(*e*) *Cases in which Ulcer is suspected.*

There are two good reasons why we should not pass a tube. In the first place, we might do mechanical injury to the stomach, and, secondly, the information which we could obtain by so doing would probably be of little value for diagnostic purposes. There might or might not be hyperchlorhydria, and blood might be present or absent.

Digitized by Google

The point which would have the most diagnostic significance would be finding small quantities of blood in the stomach, but even this we can ascertain better by the examination of the stools. The important point would be the finding blood in the stomach on many occasions, and we can quite easily see that it would be difficult to pass a stomach tube sufficiently often to render our observations of much value. But if there is blood in the stomach it must of necessity pass into the intestines, and we can make as many examinations of the stools as we wish without annoying the patient. Our observations in this direction will be of real value if we take care that the patient is fed upon blood-free articles of diet during the period occupied by our investigations. The routine which I habitually follow is to put the patient to bed and first of all give one or two doses of castor oil. The diet is then restricted to milk, white fish, bread, sweetbread, potatoes, and farinaceous substances. After the patient has lived exclusively on these for three days or so, we may be sure that the intestinal tract will now be free from any hæmoglobin derived from food. We can now commence to make a daily examination of the fæces, using for preference Teichmann's hæmin crystal test. A tiny fragment of fæces is rubbed upon a slide with a little salt. A cover-glass is applied, and some glacial acetic acid allowed to run in under its edge. The slide is then to be very slowly heated over a spirit lamp until the acid just commences to boil. If any blood be present the characteristic hæmin crystals will be seen on examination with a high power. If we find blood persistently present in the stools under the conditions mentioned above, there is in all probability an ulcer either of the stomach or duodenum.

As additional signs of some value we have the epigastric and dorsal painful pressure spots, areas of hyperæsthesia in the skin of the epigastrium, and the fact that the pain will vary inversely with the degree of comminution of the food. Also the fact pointed out by Murdock that orphol will usually relieve the pain of ulcer whilst it is without effect upon that of hyperæsthesia or gastralgia.

(*f*) *The Digestive Disturbances accompanying Cardiac Disease.*

As in the case of suspected ulcer the tube is contra-indicated, both because we may injure the patient and because we shall learn nothing of importance by passing it. The straining and general disturbance often accompanying the passage of a tube for the first time is exceedingly dangerous in organic disease of the heart, and may result in syncope or even in sudden death. The digestive troubles present in cardiac affections are in most cases either of a reflex nature or they are directly due to myasthenia, the result of venous stasis in the walls of the stomach. It is as well in all cases to make sure that they are not the result of irritation of the mucous membrane of the stomach by digitalis and other cardiac tonics. An exact knowledge of the chemistry of digestion is fortunately not very valuable in these cases, as we can do practically nothing to relieve the gastric derangement until cardiac compensation has been restored.

Such, then, are the principal cases which we are justified in treating with a provisional diagnosis, but when we come to the more difficult and obscure clinical problems no attempt should be made to prescribe until the disease has been carefully studied, and the more modern methods of examination put into operation.

(B) Cases in which Intra-Gastric Methods are necessary.

1. All cases in which there are signs of gastritis.
2. All cases which are prolonged and resist treatment.
3. All cases which are accompanied by loss of flesh, or cachexia.
4. All cases which have commenced rather suddenly in a person over middle age, who was previously in good health.
5. All cases which are evidently progressive.

In such cases the probabilities are that our diagnosis will lie between :

(*a*) Chronic progressive non-malignant stenosis of the pylorus.

(*b*) Myasthenia with retention.

(*c*) Permanent hypersecretion.

(*d*) The later stages of chronic gastritis.

(*e*) The severe form of gastric neurasthenia.

(*f*) Malignant disease.

In order to make a diagnosis we shall require certain data from which we may legitimately make deductions. If our facts have been correctly observed,

Digitized by Google

and if our logic is sound, our conclusions must be incontrovertible. The reason why the modern methods of investigating diseases of the stomach have not yet become part and parcel of the every-day routine of the rank and file of the profession is, I am afraid, on account of difficulties which are for the most part purely imaginary. As a matter of fact the tests themselves are not difficult, do not require laboratory facilities, and are by no means many in number. One simply requires to know about half a dozen things to be able to say at any rate which part of the digestive process is at fault. In any given case we want to know:

(1) *Is there any Gastritis?*

The recognition of chronic gastritis is not by any means as easy as it was formerly supposed to be, when it was the stock diagnosis in the majority of dyspeptic cases. It cannot be made by the process of exclusion, nor by an analysis of the subjective symptoms, as these present nothing absolutely characteristic. When it is the primary disease and of long standing, it may quite easily be mistaken for the severe type of gastric neurasthenia or for malignant disease.

It is always of great importance to ascertain whether gastritis is present as a complication, whatever our diagnosis may be, as it must be taken into account, both for purposes of treatment and prognosis.

The only absolute sign of gastritis is the presence in the stomach of mucus of a special kind and in abnormal quantity. Mucus in the stomach contents may be recognised by the fact that filtration takes place much slower than usual. It is quite possible to miss finding mucus in the empty stomach even when present to excess, unless the proper technique be followed. First of all the aspirator should be attached to the end of the stomach tube to make sure that the stomach is really empty. It is then disconnected and the funnel and rubber tube connected. Then lavage should be performed, the same water being allowed to run backwards and forwards between the funnel and the stomach in the manner described by me elsewhere.*

The washings are then placed in conical glasses and the mucus allowed to settle, which it will do in the form of flakes or shreds. Pathological mucus, the product of gastritis, may be distinguished from that which has been swallowed by the fact that it stains deeply with methyl green, whilst the latter is only faintly coloured. Macroscopically we can usually distinguish mucus which has been swallowed by the fact that it floats upon the surface of the washwater in globular masses, and is often of a glassy appearance. Mucus which has been formed in the stomach is always intimately mixed with food, except when it has been extracted from the empty stomach by lavage in the manner described. Mucus often clings so tightly to the walls of the stomach that it cannot be dislodged by ordinary lavage. In this case we should try the effect of the intra-gastric needle-douche before concluding that mucus is absent. The statement of some authorities that mucus may be found in any considerable quantity in healthy stomachs has been pointed out by Riegel to be erroneous, and it is more than probable that the observers were misled by not recognising the presence of gastritis in the subjects of their experiments.

2. *Is the Motor Function of the Stomach normal?*

Motor insufficiency of the stomach may be defined as a condition in which it does not empty itself into the duodenum in a reasonable time. It may be caused, as we all know, either by muscular weakness of the stomach-wall or by some obstruction to the passage of food out of the stomach. Muscular weakness of the stomach may be said to exist in three degrees. The first degree of myasthenia is simple loss of tone, and is not accompanied by motor insufficiency; that is to say, the stomach empties itself before the next meal. This may be recognised by the ease with which the stomach will inflate when an effervescing powder is given, and by the occurrence of splashing after the ingestion of two glasses of water taken when the stomach is empty. A normal stomach will not splash under these circumstances. Myasthenia of the second degree, in which the stomach does not empty itself in seven hours; and that of the third, in which it is unable to do so during the night; and likewise the motor insufficiency due to pyloric stenosis, are recognised with absolute certainty by finding food residues in the stomach at a time when it ought to be empty.

* 'Manual of Intra-gastric Technique,' London, 1903, page 91.

Digitized by Google

3. *Is the Secretion of Gastric Juice confined to the Digestive Period, or is it continuous?*

This is a most important point, as the continuous secretion of gastric juice probably points to some irritation, the source of which will require further investigation. Such an irritation may be central, and due possibly to some autotoxis, or may be a local affair caused by fermenting residues of food. The presence of hypersecretion may be readily established by aspirating the stomach before breakfast. A few c.c. of fluid, with a trace of free HCl, will be of no clinical significance; but if we find over 50 c.c., with a total acidity of at least 30, and free HCl of 10 or more, not once only, but on several different examinations, we shall be justified in assuming that hypersecretion is present.

4. *Has the Gastric Juice normal Digestive Power?*

We can tell a great deal from the naked-eye examination of the stomach contents extracted after a test meal consisting of meat and bread. We may assume that it is normal if we find that the fibres of meat have undergone considerable digestion and the bread in a very finely divided condition. In hyperchlorhydria the meat will have practically disappeared, and the residue will consist of undigested bread. In cases where there is grave alteration in the gastric juice we shall find both the meat and bread in comparatively large pieces, and evidently unacted on to any extent. We must then confirm our observation by placing a little piece of hard-boiled white of egg in some of the filtrate from the stomach contents and keeping it at the temperature of the body for a few hours. This may be conveniently done in Martindale's pocket incubator. If we find that solution is imperfect we have positive proof that the peptic power of the gastric juice is reduced. In any case we should make a chemical examination to the extent of testing the filtrate of the stomach contents with methyl violet for free HCl. In cases where pain is a marked feature we should ascertain the amount of free HCl by titration in the usual manner with the decinormal solution of sodium hydrate, using one of the well-known indicators. In other cases this will not be necessary.

5. *Is there Gastroptosis or Dilatation of the Stomach?*

These conditions we diagnose by the positions occupied by the upper and lower borders of the stomach respectively. Under no circumstances in health should we be able to make out the upper border or lesser curvature of the stomach. If we can do so there must of necessity be a downward displacement of the stomach. Similarly, the lower border of the stomach should not extend below the umbilicus six or seven hours after a meal (there being, of course, no gastroptosis). We estimate the position of the borders of the stomach—

(*a*) By careful percussion.

(*b*) By percussion and palpation after inflation with air or carbonic acid.

(*c*) By estimating the area over which splashing can be obtained.

(*d*) By the use of the gastro-electro-diaphane.

This is as far as we need go in cases where we find the gastric juice retains its peptic power and empties itself normally into the duodenum. But if we find food residues in the stomach seven hours after a meal, or find absence of free HCl or of peptic power in the gastric juice, we must proceed to the following additional tests before we shall have sufficient data from which to deduce an opinion as to the nature of the case.

6. *The Amount of HCl Deficit.*

We are not justified in assuming that the stomach is secreting no hydrochloric acid because we find none of it in the free state in the stomach contents. We shall not find free hydrochloric acid unless enough is secreted to combine with all the food and yet leave some to spare. In cases, then, where the stomach contents give a definite reaction with litmus paper, and yet show no free hydrochloric acid, we should estimate the deficit. This we can do by titrating with a decinormal solution of hydrochloric acid until congo red or dimethyl-amido-azo-benzol gives a reaction. Say, for example, that we find the total acidity 30, and that 10 c.c. of the decinormal hydrochloric acid solution are required before the reaction of free hydrochloric acid can be obtained, we know that the amount secreted is quite considerable, and that the gastric mucous membrane has by no means lost its power of secreting useful gastric juice.

7. *Is the Rennet Ferment present?*

The recognition of the presence or absence of

Digitized by Google

rennin or its zymogen is of great importance in cases where we wish to ascertain whether atrophy of the secreting glands of the stomach has occurred. As far as we know at present, the absence of these ferments is a conclusive proof of this condition. The test is performed with the greatest ease by adding a few minims of the filtrate of the stomach contents to a little milk, carefully neutralising it with an alkali, and keeping it at a temperature of 98° F. for fifteen minutes. If coagulation takes place within that time, rennin is present; if not, it is absent. In many of the cases where this test is required HCl is absent and consequently rennin, if present, will be in the form of its zymogen, and will not coagulate milk. By the addition of calcium chloride we convert the zymogen into rennin and repeat the coagulation test.

In cases where we require to go through the whole investigation the following routine may be conveniently employed:—The patient takes a mid-day meal of bread and meat, and you visit him at 7 p.m. and extract the contents of the stomach.

(*a*) The stomach may be practically empty. In this event you know that the motor functions are normal.

(*b*) You find food residues. There is motor insufficiency of the second degree. Whether this is the full measure of the insufficiency you will not know until after the examination before breakfast.

You wash out the stomach clean and allow a dinner of meat and bread to be taken.

The following morning you visit the patient again before anything to eat or drink has been taken, and again extract the stomach contents.

(*a*) The stomach may be empty. In case it was found to be empty at your examination on the preceding day before the evening meal, your opinion that the motor functions are normal will be confirmed. If, on the contrary, food residues were found, you will know that although the stomach is unable to empty itself before the next meal it can do so during the night, and we have consequently motor insufficiency of the second degree only.

(*b*) The stomach contains food residues. There is motor insufficiency of the third degree.

(*c*) The stomach contains a few c.c. of a fluid, colourless, greenish or yellowish, and neutral or slightly acid. This is compatible with health.

(*d*) The stomach contains more than 20 c.c. of a fluid containing a considerable amount of free hydrochloric acid. There is hypersecretion of gastric juice. Having extracted all the stomach contents, the opportunity should now be taken before removing the tube to inflate the stomach, and to make out and mark upon the skin of the abdomen its exact position with a blue pencil. Lavage is now to be performed, and the patient given the Ewald test breakfast. One hour after this the stomach contents are to be again extracted and examined for total acidity, free hydrochloric acid, and peptic power.

The conditions which we find after the test breakfast may be grouped as follows:

(*a*) The bread is manifestly badly digested; free hydrochloric acid in excess; total acidity raised. We have certainly hyperchlorhydria present whatever its explanation.

(*b*) Bread is well digested; total acidity and free hydrochloric acid normal. The filtrate digests egg albumen and coagulates milk. The secretory functions of the stomach are normal.

(*c*) The bread is tolerably well digested; the total acidity is low; free hydrochloric acid only a trace; mucus is present in excess, as shown by slow filtration. There is chronic gastritis.

(*d*) The bread is not digested, but is in coarse pieces; the filtrate does not digest albumen, and does not coagulate milk even after the addition of calcium chloride, and there is no free hydrochloric acid. The stomach is not secreting functionally active gastric juice, and if this condition is persistent and is observed on several subsequent examinations we know that we have either achylia gastrica, the terminal stage of a chronic gastritis with atrophy of the secreting mucous membrane, or malignant disease, which we shall be able to ascertain by further investigation.

You will by this time in all probability have made a diagnosis, except in those very difficult cases where we have to distinguish between stenosis of the pylorus of a non-malignant nature, myasthenia, severe subacute gastritis with atrophy of mucous membrane of the stomach, achylia gastrica, the grave form of nervous indigestion, and incipient carcinoma. In all these cases we may have different degrees of retention of food residues in the stomach, albeit from different causes. Although we may learn a great deal from physical methods of examination, yet we must take into

Digitized by Google

consideration in forming our diagnosis the well-known methods of evolution of these several complaints.

The history of progressive stenosis of the pylorus and of myasthenia may very much resemble each other. In both cases we may have attacks of motor insufficiency of the stomach, subsiding under rest and abstinence, and separated by periods of comparative stomachic health. We shall, however, in most cases be able to distinguish between them by the water test. We can understand the *rationale* of this test if we take into consideration the conditions severally present. In stenosis of the pylorus in the early stages during which the diagnosis presents difficulty the stomach muscles retain their normal power; in fact there is often hypertrophy. Under these circumstances any water introduced into the stomach will be ejected through the pylorus without difficulty, whilst solid food will be arrested. In myasthenia we have a normal pylorus, but the muscular walls of the stomach are weakened and sag down under the weight of the stomach contents. In the erect position of the body the stomach will be more or less incapable of raising any liquid introduced into it to a sufficient height to get rid of it into the duodenum. The test is performed by giving the patient 300 c.c. of water to drink as soon as he rises in the morning. One hour afterwards he drinks an additional 100 c.c. containing 1 per cent. of glucose. The contents of the stomach are well mixed by slight massage, and a few ounces or so extracted. This portion is now tested quantitatively for sugar. It is obvious that if the proportion of glucose is still 1 per cent. the whole of the first quantity of water must have passed out of the stomach. The exact proportion remaining in the stomach in any case may be found by the formula—

100 ÷ per cent. of sugar — 100 = number of c.c. of the original 300 c.c. of water remaining in the stomach.

If the greater part of the first 300 c.c. of water has passed out of the stomach the diagnosis is pyloric stenosis. If the greater part remains in the stomach the patient is suffering from myasthenia of a severe type. Of course in the later stages of pyloric stenosis where myasthenia has supervened this test would be useless, but in these cases the diagnosis would be sufficiently obvious.

The sort of case which gives us the most trouble to diagnose is one which presents the following characteristics:

The patient, a man of middle age, has suffered from indigestion for the last few months. There has not been any considerable loss of flesh, but he continues to get worse. Such a case is very common. We give him a test meal, and find that the stomach empties itself about three hours afterwards. If we aspirate a little before this period we find that the stomach contains undigested food in coarse particles covered with mucus. The filtrate of the gastric juice does not digest white of egg in a waterbath, contains a little lactic acid, but no free hydrochloric. We are at this point justified in saying that the motor functions of the stomach are normal, but that the peptic power of the gastric juice is much reduced. The diagnosis will probably rest between malignant disease in an early stage and atrophic gastritis, although there are other possibilities which space will not now allow us to discuss. Let us see whether we can learn anything more by extending our investigations. But first of all as to probabilities, taking the history into consideration. Up to the last few months the patient has not suffered with his stomach. This is a most important point, for if the disease were atrophic gastritis it could only be the terminal stage of a dyspepsia which had extended over many years. It is consequently highly improbable that this is a case of chronic gastritis We renew our examination of the stomach contents and ascertain that whilst rennin, as such, is absent, the rennin zymogen is present in considerable quantity. This fact, and the absence of mucus in excess, will practically exclude atrophic gastritis.

The task now before us is to confirm our suspicion that the patient is suffering from the early stage of malignant disease. This is one of the most difficult problems in medicine, and also one of the most important, since the only chance which there is of arresting the disease depends upon its recognition at a very early stage. By the time the classical symptoms are apparent it will probably be too late. As Riegel appositely remarks*: "If all the important symptoms as tumour, advanced cachexia, abundant quantities of lactic acid and absence of free hydrochloric acid, coffee-ground

* 'Diseases of the Stomach,' Philadelphia, 1903, page 701.

Digitized by Google

vomit, etc., are present, the diagnosis is easy, but it has about the same significance, as far as the patient is concerned, as a post-mortem diagnosis."

In a case like the imaginary one which we are discussing the next thing to do is to ascertain whether lactic acid is produced in the stomach under the only condition in which it is of diagnostic value—namely, after a meal free from lactic acid germs has been introduced into a perfectly clean stomach, a stomach as clean as we can make it by ordinary lavage. Such a meal will consist of a porridge made with oatmeal which has been sterilized in an oven shortly before use. If after such a breakfast any lactic acid is found in the stomach contents, it proves almost conclusively that a portion of the stomach wall has lost its motility, and has thus given a colony of lactic acid bacilli an opportunity to form what practically amounts to a plate cultivation upon it. The adherent mucus in which the colony lives cannot be dislodged by ordinary lavage, and upon the introduction of any farinaceous material into the stomach will promptly set up the lactic acid fermentation in it. As malignant disease is practically the only condition which we are likely to meet which will produce such a local infiltration of the stomach wall, if we find lactic acid the certainty of our diagnosis is materially increased.

Another point which will help us is to examine the washings from the fasting stomach microscopically. If we find nuclei of cells undergoing atypical mitosis the probability of malignant disease will be very strong.

I will conclude this brief sketch by expressing my firm belief that in the majority of these obscure and complicated cases a diagnosis can usually be arrived at by a patient and systematic investigation, first of all collecting absolute facts, and then making such deductions from them as we may be justly entitled to.

Harley Street,
March 31st.

PERSSE, LTD., Distillers, of Galway and 1, New London Street, London, have submitted a sample of their ten years' old pure unblended pot-still whiskey for notice. It is excellent whiskey, and particularly well suited for prescribing in those cases where the medicinal dose must be depended on for the therapeutic effect desired. Its claim to purity is fully justified by the examination to which it has been subjected.

WITH DR. CAMPBELL THOMSON AT MIDDLESEX HOSPITAL.

CLINICAL STUDIES IN DISEASES OF THE NERVOUS SYSTEM.*

A CASE OF INJURY TO THE CEREBRAL CORTEX.

GENTLEMEN,—You can see from the way in which this patient walks that he is suffering from some degree of hemiplegia on the left side. His left leg drags along the ground, and in order to try to clear it he swings it outwards by rotating it at the hip-joint. You can at the same time see that his left arm is more or less helpless.

His story is a very interesting one. He tells us that in 1899, in the early days of the South African war, he was wounded by a fragment of shell on the right side of the head, and was unconscious for some hours. On the following day he was trephined, after which he made a rapid recovery, felt quite well, and went to serve at the front again. At the end of about five months, however, he began to suffer from "dizzy turns," during which everything seemed to go round. These would occur two or three times in a week, and at last one day when he was mounting his horse he fell right over the other side. In consequence of these symptoms he was sent into hospital, and was again trephined in 1900, and he says it was then thought that the bone which had been replaced after the previous operation had become displaced, and it was therefore removed altogether. This operation was followed by some loss of power and numbness in the left side for a time; but the loss of sensation was apparently never very complete, and the power gradually returned until he could grip almost equally well with both hands, and in walking he only felt a difficulty when going up-hill. But unfortunately, in spite of this second operation, the fits not only continued but became more severe. They began in the left thumb and hand, and from there the movements spread up the arm, and on reaching the shoulder they passed into the left side of the trunk, after which consciousness

* These "Studies" are from the teaching in the Out-patient Department of Middlesex Hospital.

Digitized by Google

was frequently though not always lost. At this time the fits occurred every three or four weeks, and he would sometimes have as many as eight or nine in a night. At present the fits are occurring more often, viz. about once a week, so that you see the tendency is for them to get worse. Three months after the second operation a third attempt was made to relieve him, but with no permanent benefit.

From this interesting history the sequence of events can be traced fairly clearly. The injury by the shell was, in spite of the first operation, evidently followed by some permanent change in the brain tissue, and this gave rise to the epileptiform attacks, which showed themselves by the dizzy feelings, one of which finally caused him to fall off his horse. On the strength of this, it was thought advisable to trephine him again, as it was doubtless considered that there might be some removable condition giving rise to irritation, and, according to what the man tells us, it was supposed at the time of this operation that the circular piece of bone which had been cut out at the former operation had become displaced, and this time it was accordingly taken away altogether. This second operation was followed by some interference with voluntary power, which, however, gradually passed off, but unfortunately the fits still persisted, and in three months' time yet another attempt was made to get rid of them, but again with no success.

All this took place in 1899 and 1900, and he is still having fits, which are more frequent and more severe than they were formerly. We can therefore say that the irritation which first caused the fits still persists, and we can go further and say that, owing to the manner in which the fits begin, by contractions of the muscles of the hand, the chief centre of irritation is situated in that part of the cerebral cortex in which the movements for the hand are situated, and that the part of this area which has suffered most is that in which the movements of the thumb are represented. These fits in fact belong to the variety known as Jacksonian epilepsy, so called after Dr. Hughlings Jackson, who first recognised the relationship between their local origin and the situation of the lesion in the brain which caused them. The chief characters of such fits are that the movements begin in certain groups of muscles and then spread along definite lines corresponding to the arrangement of the motor centres in the cerebral cortex. These centres are, as you know, arranged in the order of leg, arm, face, from above downwards, in that part of the cortex which is in the region of the fissure of Rolando. From experiments on various kinds of monkeys it has until lately been considered that these centres are in man situated on either side of the fissure of Rolando, that is, in the ascending frontal and ascending parietal convolutions; but the more recent experimental researches of Professor C. S. Sherrington and Dr. A. F. Grünbaum * show that in the anthropoid apes, and therefore most probably in man, these motor centres only exist in front of the fissure of Rolando; that is, in the ascending frontal and not in the ascending parietal convolution (*v.* Fig.).

You will also notice, if you look at this figure, another very important point in regard to the position of the centre for movements of the trunk. This centre has hitherto been placed on the edge of, and overlapping, the superior longitudinal fissure in front of, and rather above, that for the leg; but you will see that, according to Professor Sherrington and Dr. Grünbaum, it is situated between the arm and leg centres. Now, in this connection there is a point of special interest in the case we are at present considering; for, if you remember, the patient has told us that, when he has the fits, the movements, after passing from the hand up to the shoulder, appear to go straight into the left half of the trunk. Now, if you look at the diagram you will see that, according to the conclusions of Professor Sherrington and Dr. Grünbaum, this is exactly what might be expected, for if the nervous discharge begins in the centre for the hand and then passes upwards to the centre for the shoulder, it will naturally, according to the ordinary course of events, affect the trunk next; whereas, according to previous localisations, the discharge would have been more likely to pass through the leg centre before it reached that of the trunk.†

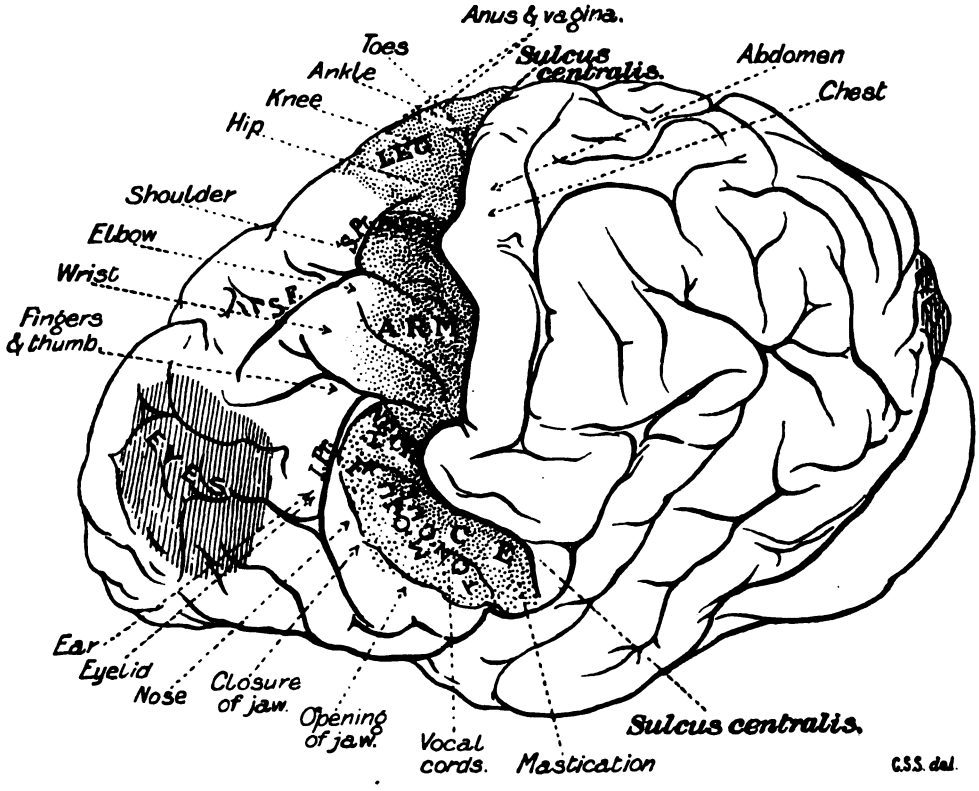
* 'Transactions of Pathological Society of London,' vol. liii, 1902.

† The patient was questioned very closely, but was always quite consistent; and from what he said and from what could be gathered from the fits which he had while in hospital, there seemed no reason to doubt that the movements passed from the arm to the trunk.

Digitized by Google

On examining the patient we find that there is considerable weakness of the left arm and leg, and also to a slight extent of the left side of the face. There is some rigidity and contraction of the fingers and thumb on the affected side, so that they are flexed tightly into the palm, and considerable force is required to extend them. This confirms what has already been said with regard to the irritation chiefly affecting the arm centre.

On coming to the reflexes it is found that the knee-jerks are increased on both sides, but rather regard to ultimate prognosis. There are also sensory changes present. There is loss of sensation to touch, pain, and temperature to a considerable degree over the affected arm and leg, and also, to some extent, over the left side of the trunk. These changes are important, since there is a considerable difference of opinion as to whether the so-called motor areas possess sensory functions as well. It is, of course, a fact that diseases of the motor cortex are often accompanied by sensory changes, but it is still an open



Arrangement of motor areas in the brain of a chimpanzee (Sherrington and Grünbaum). [I am indebted to the kindness of Professor C. S. Sherrington for permission to reproduce this figure.]

more so on the left. The behaviour of the plantar reflexes is interesting. On the right side there is a brisk flexor response, while on the left (that is, the hemiplegic side) there is only a slight movement of the foot on the ankle, and no movement of the toes at all. There is no ankle clonus on either side.

On the whole, then, the reflexes do not suggest that any serious amount of degeneration has yet taken place in the motor fibres coming from the injured cortex, a point of some importance in question as to how far these disturbances are directly due to changes in the motor area of the cortex, or how far they are due to indirect effects on fibres at a distance. In this case the distribution of the sensory changes very closely corresponds to that of the paresis, but from what has just been said it will not do to assume offhand from this that both motor and sensory changes are due to a lesion of one and the same part of the cortex.

Next, the nature and position of the lesion have

Digitized by Google

to be considered. Taking the distribution of the weakness together with the convulsive attacks, there can be no hesitation in placing the lesion in the motor region of the cerebral cortex, and we can go further and say that the lesion has given rise to both destruction, as shown by the paresis, and to irritation, as shown by the epilepsy; and moreover, we can say that while the area of the destructive lesion is sufficiently widely spread to involve the movements for the leg and arm, it is chiefly the hand area that has suffered from the irritation.

As to the nature of the lesion, it seems to me to be most likely that, considering the extent of the cortex which must be involved, it in all probability consists of a thickening and adhesion between the dura mater and the cerebral cortex, and on this supposition the patient will be advised to consent to undergo yet another operation.

One would hope by this means to set free any adhesions, and especially to remove the source of irritation. To do this might possibly entail some permanent paralysis of the hand, but the limb, contracted as it is now, is quite useless under the circumstances, and in addition to this there are the fits which are making the patient's life practically useless.

[Mr. T. H. Kellock kindly saw the patient with me, and agreeing that relief might be obtained, he operated.

An incision was made round the scar of the former operations, and on exposing the surface of brain underneath a marked thickening of the dura mater was found over the motor area. This thickened membrane was adherent to the brain tissue; but it was carefully separated, and the ascending frontal and parietal lobes were exposed to view. It was then found that there was a considerable amount of scar tissue extending into the cerebral cortex along the course of the fissure of Rolando, and this was, as far as possible, removed until apparently healthy brain tissue was exposed. In order to prevent adhesions reforming, a piece of gold leaf was inserted between the brain and soft parts, so as to completely cover the area of brain exposed. The patient was greatly improved by the operation, the rigidity of the hand almost disappeared, power improved, and the fits ceased. Up to the end of October, 1903, he had not had a single fit since the operation, and the only symptom was some weakness of the arm and leg, which he considered was not nearly so marked as before the operation.]

FOUR LECTURES
ON
NORMAL AND ABNORMAL CONDITIONS OF THE BLOOD.*

By EDMUND CAUTLEY, M.D.Cantab., F.R.C.P.Lond.,

Physician to the Belgrave Hospital for Children and the Metropolitan Hospital.

LECTURE IV.
THE THYMUS GLAND.

GENTLEMEN,—The thymus is a ductless gland, characteristic of all true vertebrates, and demonstrated by Schaffer in marsipobranchiate fishes. In colour it varies from a reddish-grey to dark red. It is situated in the anterior mediastinum just above the pericardium, and extends upwards to about 2 cm. above the sternum and 2 cm. below the thyroid, lying between the innominate and left common carotid arteries. Behind the manubrium, where it is thickest, it covers part of the vena cava superior, the innominate veins, and the arch of the aorta, and extends backwards to the trachea. In transverse section in the infant it may occupy as large an area as that of the left lung. Sometimes it extends more to the left than the right, and may cause marked dulness on percussion. The dulness is continuous with the cardiac dulness. It receives its blood-supply from the inferior thyroid arteries and its nerve-supply from the sympathetic.

Structurally it is somewhat flat, and consists of two lobes, which are not usually symmetrical, joined by an isthmus. It is covered by a fibrous capsule, from which bands of fibrous tissue pass into its substance, subdividing it into lobules. The blood-vessels and lymphatics are carried by these fibrous bands. The lobules are further subdivided into follicles by connective tissue.

Microscopically each follicle is seen to be more or less polyhedral in shape, and to consist of a denser cortical and looser medullary portion, the adenoid tissue of the cortex being more densely filled with lymphoid corpuscles. In the medullary portion are seen the concentric corpuscles of Hassall, protoplasmic masses consisting of a nucleated

* Delivered at the Medical Graduates' College and Polyclinic.

Digitized by Google

granular centre surrounded by flattened nucleated cells. They are the remains of epithelial cells.

Measurements.

	Vary up to	Sappey.	Marfan.	Brouardel.*
Length .	10 cm.	2 ins.	Up to 3 ins.	3½ ins.
Breadth .	5 „	⅘ „	„ 1½ „	1½ „
Thickness .	1 „	⅖ „	„ ⅘ „	⅘ „

Observations on weight.—It is usually largest shortly after birth, and after the age of two years gradually diminishes. Although it may weigh as much at puberty as in infancy, its structure is altered, and it chiefly consists of fibrous tissue. In young adults it may weigh as much as 22 grms. Probably, although it undergoes involution, it never entirely disappears. Its weight is given differently by various observers, and no doubt varies very greatly in normal infants and still more so in those suffering with some one or other of the numerous ailments of early life. Naturally the observations have been largely made on infants who have died from some cause or other which has caused a departure from health. Except in the cases of death from accident and a few other causes, we must not accept the weight as a normal one without careful consideration.

Weight of the fœtal thymus.—Dudgeon examined ten cases from four months up to term, and found that the weight varied from 0·6 grms. to 22·2 grms., the average being 4·57 grms. The very large thymus was found in an anencephalic fœtus, and is usual in such monstrosities. In structure the fœtal gland resembles that of early infancy. Many eosinophiles are present. The differentiation between cortex and medulla is ill marked at the fifth month.

At birth.—The gland weighs on an average 13 grms. (Friedleben), 11 to 13 grms. (Mettenheimer), 5 grms. (Testut).

In infancy.—Katz gives the average weight in twenty babies under six months as 11 grms., in twenty-five babies aged from six months to two years as 6 grms., and in eighteen over the age of two years as 8 grms. His figures are much smaller than those of Friedleben, who gives 19 grms. as the average weight at nine months and 26 grms. at two years. Mettenheimer states that the average weight under two years is 18 grms., and so do Stokes, Rurhräh, and Röhrer in their joint paper. On the other hand, smaller weights are given by Sappey, 3 to 4 grms.; and, as an average in children under one and a half years of age, 6 to 7 grms. (Thursfield), 7·1 grms. (Dudgeon). Marfan states that it may weigh up to 30 grms.

Development.—The place of origin and mode of development are the same in all vertebrate animals (Kölliker), and agree essentially with those in the smooth skate, *Raia batis*, a vertebrate with a plan of organisation which is practically the same as that of man (Beard). The minute anatomy is also the same, with the exception that Hassall's corpuscles have not been found in the skate. In the thymus of dogs and some other animals cysts are found, lined with ciliated or short columnar epithelium, and probably derived from the corpuscles of Hassall. The gland is hypoblastic in origin, being developed from the epithelium of the third pouch of the gut-tract.

Functions.—Stieda and His (supported by Dohrn, Gulland, Möhrer) state that the functions of the gland are absolutely unknown; that Hassall's corpuscles arise from the original epithelial cells; and that the leucocytes migrate into the gland from the exterior, possibly from the mesoblast. Watney states that the gland takes part in producing erythrocytes from its colourless cells in early life. Chiari and Ziegler regard it as the lymphatic system of early life. Certainly it appears to be intimately connected with the lymphatic system. Kölliker puts forward the view that the gland is the parent source of leucocytes or lymphocytes, by transformation of the epithelial cells. His views are supported by Prenant, Oscar Schultze, and Beard. Some important and interesting observations on the skate have been brought forward to assist in the solution of the problem. Both Kölliker and Beard found that for a relatively long period of development the blood only contained nucleated red corpuscles and no leucocytes. H. E. Ziegler suggested that this condition persisted until some lymphoid organ was formed. Now, at the period when leucocytes first appear there is no spleen, rectal gland, or cæcum, while the thymus gland is represented by small pieces of modified

* A case of sudden death in a child æt. 3½ months.

Digitized by Google

epithelium, five on each side. Beard suggests that the first leucocytes are formed from these epithelial cells, a conversion analogous to that of the epithelial cells of the brain into nerve-cells. This absence of leucocytes from the blood persists until the first ones are formed from the thymus epithelium. At first they are formed singly, later on in crowds. At this period there are no leucocytes or lymphoid tissue in any other part of the body. Hence the thymus may be regarded as the parent of all leucocytes, and it alone is sufficient to account for them all. The blood and tissues become infiltrated with these leucocytes before lymphoid tissue is found elsewhere.

Experimental results.—Friedleben found that extirpation of the gland in dogs caused changes in the blood; disturbances of growth, more especially in the bones; nervous symptoms, and profuse sweating. This is a constitutional state resembling rickets, and the gland has been prescribed in the treatment of this disease.

Hypertrophy of the gland is found in the condition described by Paltauf as the *status thymicus*, in the thymic asthma of infants, in many cases of sudden or unexpected death in infancy, in leukæmia and Hodgkin's disease, in about 43 per cent. of cases of acromegaly, and in some cases of gigantism, chlorosis, exophthalmic goitre, and epilepsy. In thirteen cases of diphtheria Katz found it was heavier than he had found it in normal children, weighing up to 18 grms. Stokes, Rurhräh, and Röhrer give the average weight in seven cases of diphtheria as 14·35 grms., less than what they regarded as normal.

Atrophy, or absence of the gland, is found in various conditions. Thus it may be absent in the mentally deficient. Bourneville found no trace of its presence in twenty-five out of twenty-eight imbecile, idiotic, epileptic, and mentally deficient children, and states that it was absent in 73 per cent. of 292 mentally abnormal children. Katz found it present in sixty-one mentally sound children under thirteen years of age, forty-one of them under two years of age, dying from various diseases. Various observers have found that it is atrophied in infantile atrophy or marasmus, and, indeed, in any chronic wasting disease. The weight of the gland is the best index of the state of nutrition of the body in infancy. It is atrophied in rickets and in atrophic conditions of lymphoid tissues. According to Friedleben, it diminishes in size as the spleen enlarges. Schaffer states that it contains few nucleated cells if there are many in the spleen, and *vice versâ*.

Primary atrophy of the thymus in association with marasmus.—Valuable observations on this subject have been recorded by Dudgeon and by Stokes, Rurhräh, and Röhrer. The gland is soft, white, œdematous, and cuts with great resistance. No pus-like fluid is exuded on pressure. In weight it varies, but is almost always much diminished.

Weight in Marasmus.

Observer.	Weight.	Limits.	No. of cases.
Durante	1·5 grms.		
Farret	1·6 „		
Stokes, etc.	2·2 „	1·2—7·5 grms.	16
Dudgeon	2·68 „	0·905—5·15 „	15
Mettenheimer	2·85 „	2·1—3·6 „	2

Except in cases of terminal infection, the changes in the gland were the only ones found, and the mucous membrane of the intestinal tract was usually well preserved.

Microscopical examination of the gland enables us to form a good estimate of the state of nutrition of the infant. If the nutrition be normal, the gland is unaltered, and the differentiation between cortex and medulla is distinct. In moderate atrophy the cortex and medulla are not easily distinguished. In severe marasmus marked changes are found. The fibrous capsule and trabeculæ are greatly thickened, cutting up the lobules into irregular masses. The thickening is especially marked about the blood-vessels. Sometimes the fibrosis is so marked that, under a low magnification, more fibrous tissue than lymphoid is visible. The normal lymphoid structure is greatly altered. The endothelial cells lining the reticular spaces and in the reticular tissue have undergone proliferation, and largely replace the lymphocytes, which are consequently decreased in number. Small giant-cells, somewhat like those seen in the lymph-glands in lymphadenoma, are found (Dudgeon). Sometimes the gland is infiltrated with fat-cells.

Digitized by Google

According to Stokes, Rurhräh, and Röhrer, the most striking feature is the increase in size and the degeneration, usually hyaline, of Hassall's corpuscles. Van Gieson's fluid stains them rose-red if they are converted into hyalin, and orange if they are converted into kerato-hyalin. The greater the atrophy of the gland, the greater is the conversion into true hyalin. Dudgeon, using this stain, found that the corpuscles were all stained bright yellow, occasionally orange. Possibly the different results are due to the use of different mixtures of the staining fluid. Sometimes the corpuscles are homogeneous, granular, and composed of compressed scales, calcareous, or converted into cysts. More often they are infiltrated with polynuclear leucocytes.

Secondary atrophy of the thymus.—No special changes are found in cases of acute illness. In chronic ailments, such as tuberculosis, rickets, congenital syphilis, and gastro-enteritis, Mettenheimer found decrease in weight, fibrosis, and hyaline degeneration. Stokes, etc., found considerable decrease in weight, atrophy of the lobules, increase in the interlobular connective tissue, obliteration of lymphoid spaces by proliferation of the endothelial cells, endarteritis, loss of the normal structure of the gland, decrease in the number of lymphocytes, and degeneration of Hassall's corpuscles. Dudgeon found that fibrosis was the most marked change. In several the gland was infiltrated with adipose tissue. In thirteen of his cases there was no tuberculosis; in four there was extensive caseation of the gland. The average weight was 3.065 grms., the minimum being 0·0345 grms. Stokes, etc. found the average weight in thirteen cases of miliary tuberculosis, congenital syphilis, and gastro-enteritis was 4·5 grms. In eleven cases of scarlet fever it averaged 12·05 grms.—less than the 18 grms. which they regard as the normal average weight, but equal to or greater than that given by some other writers.

Status thymicus.—Under this name Paltauf described as a morbid entity a condition of hyperplasia of the thymus and lymphatic apparatus generally, including the spleen. With this there is associated an abnormal predisposition to sudden or unexpected death in infants and adults. In a few cases in adults it has been found associated with hypoplasia of the heart and aorta (co-arctation). There is great liability to death under anæsthesia or from shocks, such as a cold plunge; or even from injection of antitoxin, as in the celebrated case of Professor Langerhan's child.

The signs of a thymic constitution are indefinite. Many patients seem to enjoy perfect health. Usually the complexion is pale, the skin thin and pasty. In addition to the enlarged thymus, there may be found rickets, enlarged cervical and axillary glands, hypertrophy of the circumvallate papillæ, of the adenoid tissue in the pharynx and the tonsils, and enlargement of the spleen.

The mode of death varies. It may be due to collapse on immersion in cold water, to slight convulsions, or to thymic asthma, an asphyxia of obscure nature which must not be confounded with laryngismus stridulus. It is sometimes ascribed to œdema of the brain. The pathology is most obscure. Possibly it is a constitutional state dependent on faulty nutrition. Or death may be due to a toxæmia, occurring intermittently, as a result of an abnormal state of the lymphatic system.

Thymic asthma and sudden death.—A congenitally large thymus may give rise, by partial compression of the trachea or in other ways, to dyspnœa, stridor, aphonia, and even sudden death. The name of thymic asthma was given to this condition by Millar. Grawitz noted that children with an abnormally large gland might die suddenly. Hérard, in 1847, and Bontemps, in 1882, reported similar cases. The following cases are illustrative of the affection:

1. Infant, æt. 3½ months. Taken ill with dyspnœa, cyanosis, and distress. Sudden death at the end of nine hours. The thymus was much enlarged, and so, too, were the glands in the mediastinum, mesentery, and groins. The spleen and liver were enlarged. Death was due to pressure on the great vessels or possibly to the status thymicus. (E. C. Jones.)

2. Boy, æt. 3½ months. Subject to attacks of "grunting and lividity." He was unusually big and fat. Breathing was normally laboured, with dyspnœa and cyanosis in the attacks. Death resulted during an attack three weeks later. Tracheotomy proved useless. The thymus was found to be large. Death was ascribed to pressure on the trachea or recurrent laryngeal nerve. (J. E. S. Barnett.)

3. Infant, æt. 6 months, with loud systolic murmur over the base of the heart, convulsive

Digitized by Google

seizures twice, and sudden death. The large thymus was supposed to have compressed the trachea. (A. Caillé.)

4. Boy, æt. 7 months. He was supposed healthy, but woke up from a sound sleep, sat up, became blue, and fell back dead. The thymus covered the front of the pericardium and reached the thyroid. It was two and a half inches square and three-quarters of an inch thick. (Köppe.)

5. Infant, æt. 10 months. The symptoms were those of croup, with projection of the manubrium and impaired note over it. Intubation and tracheotomy were both useless. The thymus was large and pressed on the upper part of the trachea and sternum. (Biedert.)

6. Boy, æt. 2½ years. He had suffered for two months from attacks of suffocation with cyanosis and recession. Tracheotomy was useless until a tube was inserted, reaching the bifurcation of the trachea. Removal of the tube induced a fresh attack. The child was cured by removal of part of the gland. The symptoms in this case might easily have been mistaken for those of laryngismus stridulus. (Siegel.)

Dudgeon examined the gland in sixteen cases of "sudden death" and "found dead." The average weight was 25 grms., varying from a minimum of 7 grms. 770 mgrms. in a child of sixteen months to a maximum of 47 grms. 50 mgrms. in a child of five months. On cutting and pressing a large gland thick, yellowish-white, pus-like fluid appears. If post-mortem degeneration was advanced, regular cavities filled with this fluid were found. This fluid has been mistaken by some observers for pus. It contains no pus-cells, and consists almost entirely of leucocytes. It is due to liquefaction of normal lymphoid tissue, and is not pathological. It is sterile, unless due to secondary infection. The other post-mortem changes found were well-marked rickets in most cases; hypertrophy of the lymphoid tissue of the pharynx; in three cases enlargement of the spleen, glands, solitary follicles, and Peyer's patches; in two cases superficial hæmorrhages in the thymus; subpleural and pericardial hæmorrhages in several; and an enlarged thyroid in one. No cases showed thrombosis of arteries or veins, distension of the right side of the heart with blood-clot, or narrowing or twisting of the trachea. Microscopically there were found large cells containing eosinophile granules, especially numerous along the septa and in the connective tissue round the small blood-vessels. He found four varieties of these cells, and regarded them as characteristic of hyperplasia of the thymus.

Symptoms.—The physical signs of enlarged thymus are dulness and the effects of pressure—on the trachea, causing dyspnœa and spasm; on the right ventricle and the pulmonary artery, interfering with the passage of the blood through the lungs; and on the vena cava superior or the innominate veins, interfering with the return of blood to the right heart; and on the nerves, giving rise to attacks of spasm or thymic asthma. The dulness, due to the thymus, is over the manubrium. It is roughly triangular in shape, with the base upwards and a somewhat cone-shaped apex at a level with the second rib. It extends a little beyond the manubrium on either side, more to the left than the right. When enlarged, the dulness is continuous with that of the heart. It may be present up to the end of the fifth year, but is inconstant after that age. Light percussion is necessary to elicit it.

Usually the child appears to be in good health, but while suckling, or drinking out of a cup or spoon, the head is thrown back, and death occurs without a cry or convulsion, or even any movement at all. Probably such sudden death is due to spasm of the glottis. Dyspnœa and cyanosis are indications of pressure. Stridor and aphonia have also been noted; so, too, a systolic murmur over the base of the heart. The asthmatic attacks are often mistaken for laryngismus stridulus. As a rule they differ from the latter affection in the absence of the characteristic "crowing" and the presence of regular "sighing" respiration throughout the attack.

On post-mortem examination no evidence of compression of the trachea has been found, perhaps because the trachea is very elastic and resumes its normal position when the sternum is removed.

There are several probable causes of death in these cases: (1) laryngo-spasm; (2) pressure causing asphxyia—of this there is often no evidence; (3) status thymicus, and the action of a toxin; (4) intra-vascular clotting—of this Dudgeon found no evidence. Wooldridge has shown that an extract of thymus injected into the veins will cause intra-vascular clotting.

The *diagnosis* of enlargement of the thymus

Digitized by Google

depends mainly on the evidence obtained by percussion. It may be confirmed by physical signs due to the effects of pressure, such as dyspnœa, cyanosis, thymic asthma, and the presence of a systolic cardiac murmur. Often in cases of sudden or unexpected death there have been no symptoms to attract the attention, previously to the fatal issue. We must not put down all cases of sudden death to this cause. Thus, it may occur from various respiratory conditions, among the chief of which may be mentioned (1) coryza in the new-born, giving rise to aspiration and the so-called "swallowing of the tongue," the tongue being drawn upwards and backwards, preventing the entrance of air; (2) pertussis, acting in a similar manner; (3) asphyxia from overlaying; (4) spasm of the glottis, due to irritability of the nervous system in rickets and debility, or independent of either. Some of these deaths are really due to syncope.

Treatment.—It is unlikely that any good can be done, except by operation in very severe cases. If the symptoms are due to pressure, intubation is useless and tracheotomy of only temporary benefit, even if a long tube be inserted. The only chance of permanent benefit lies in the partial or entire removal of the gland. König has recorded a case in a child, æt. 2 months, who had suffered from dyspnœa since the eighth day. The gland was drawn up and fixed so that it could not press on the trachea or great vessels, and the suffocative symptoms were relieved. In Siegel's case recovery followed the partial removal of the gland. In a case of Perrucker's, æt. 2½ years, the entire gland was removed for the cure of dyspnœa, stridor, and aphonia, which had existed since the age of fifteen days. No ill effects were noted eight months later. It does not follow that in all cases in which a diagnosis of enlarged thymus is made the gland should be removed. Careful consideration must be given to the severity of the symptoms, for many infants present such symptoms and gradually improve. On the other hand, it must not be forgotten that sudden death may occur even in the absence of symptoms.

Other affections of the thymus.—Miliary tubercles are sometimes found, and, less frequently, small tuberculous nodules. Primary inflammation has been recorded. Multiple minute hæmorrhages have been found in asphyxia neonatorum, and scattered hæmorrhages are present in almost all affections of the lungs. Dubois stated that he found multiple abscesses in several cases of congenital syphilis. Chiari regarded them as necrotic areas or cysts. These are not abscesses, but are simply due to softening of normal lymphoid tissue, as described above. Gummata are very rare.

In a man, æt. 30 years, who died after four to five years' illness from myasthenia gravis, Laquer and Weigert found a tumour in the region of the thymus, composed of small-cells, lymphoid cells, epithelioid cells, and Hassall's corpuscles. The tumour had grown into both veins and arteries. The muscles were invaded by the small-cells, which are not normally found in the thymus, and to a less extent by the epithelioid cells. The muscle-fibres were normal. They gave the myasthenic reaction during life. Similar foci of cells were found in the heart muscle. This is the first time any pathological changes have been demonstrated in this interesting disease.

Therapeutic value.—Mendel dosed 100 rachitic children with thymus gland, and attributed improvement to the treatment. It is said to relieve the nervous symptoms. It has proved of benefit in some cases of myxœdema. Stokes found it of no benefit in marasmus in doses of one to three grains three times a day.

Bibliography.

1. Barnett.—'Lancet,' 1898, vol. i, p. 1185.
2. Beard.—'Lancet,' 1899, vol. i, p. 144.
3. Biedert.—'Brit. Med. Journ.,' Epitome, 1896, vol. ii, No. 100.
4. Caillé.—'Archiv. of Pediatrics,' March, 1903.
5. Dudgeon.—'Path. Soc. Trans.,' Lond., 1904; 'Brit. Med. Journ.,' 1903, vol. ii, p. 1533.
6. Friedleben.—'Monograph on Eight Years' Investigations of Three Hundred Cases,' 1858.
7. Griffith.—'Amer. Med.,' June 20th, 1903.
8. Jones.—'Archiv. of Pediatrics,' August, 1903.
9. Katz.—'Le Progrés Medicale,' June 23rd, 1903.
10. Köppe and Siegel.—Vide 'Rev. des Mal. de l'Enf.,' May, 1897.
11. Kölliker.—'Entwicklunge-geschichte des Menschen und der höheren Thiere,' 1879, pp. 875—880.
12. Laquer and Weigert.—'Neurol. Centralb.,' July, 1901; and 'Lancet,' 1901, vol. ii, p. 158.
13. Mendel.—'Münch. med. Woch.,' January, 1902.
14. Stokes, Rurhräh, and Röhrer.—'Amer. Journ. Med. Sciences,' 1902, vol. cxxiv, p. 847.
15. Testut.—Vide 'Quain's Anatomy.'
16. Thursfield.—'St. Bartholomew's Hospital Reports,' 1902, vol. xxxviii.

Digitized by Google

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

EDITED BY L. ELIOT CREASY.

No. 598. WEDNESDAY, APRIL 13, 1904. Vol. XXIII. No. 26.

CONTENTS.

PAGE

*What is Syphilitic Disease of the Nervous System? By GEORGE OGILVIE, B.Sc., M.B., M.R.C.P. 401

*On the Treatment of Appendicitis. By W. H. B. BROOK, M.D., F.R.C.S. ... 413

* *Specially reported for the Clinical Journal. Revised by the Author.*

ALL RIGHTS RESERVED.

NOTICE.

Editorial correspondence, books for review, &c., should be addressed to the Editor, 35a, Welbeck Street, Cavendish Square, W., Telephone No. 904, *Paddington; but all business communications should be addressed to the Publishers, 22½, Bartholomew Close, London, E.C. Telephone* 927, *Holborn.*

All inquiries respecting Advertisements should be sent to Messrs. Adlard & Son, Bartholomew Close, E.C. Telephone 927, *Holborn.*

Terms of Subscription, including postage, payable by cheque, postal or banker's order (in advance): for the United Kingdom, 15*s.* 6*d. per annum; Abroad,* 17*s.* 6*d.*

Cheques, &c., should be made payable to THE PROPRIETORS OF THE CLINICAL JOURNAL, *crossed "The London, City, and Midland Bank, Ltd., Newgate Street Branch, E.C. Account of the Medical Publishing Company, Limited."*

Reading Cases to hold Twenty-six numbers of THE CLINICAL JOURNAL *can be supplied at* 2*s.* 3*d. each, or will be forwarded post free on receipt of* 2*s.* 6*d.; and also Cases for binding Volumes at* 1*s. each, or post free on receipt of* 1*s.* 3*d., from the Publishers,* 22½ *Bartholomew Close, London, E.C.*

The Index and Title-page for Vol. XXIII will be published with the next issue of THE CLINICAL JOURNAL.

WHAT IS SYPHILITIC DISEASE OF THE NERVOUS SYSTEM?*

By GEORGE OGILVIE, B.Sc., M.B.Edin., M.R.C.P.Lond.,

Senior Physician to the Hospital for Epilepsy and Paralysis, and Physician to the French Hospital, London.

GENTLEMEN,—I am not asking your attention for an enumeration of the many pathological conditions of which syphilis may be the cause, nor for a description of the manifold manifestations by which this protean disease reveals its existence, even after long periods of latency. Such a task would far exceed the limits of a single lecture, and is perhaps better left to the text-book writer. My purpose is to consider the conditions under which we are justified in assuming the syphilitic origin of a disease, and to point out some of the difficulties and the principal sources of error in diagnosis. Here, again, I shall limit myself to affections of the nervous system. Of course much of what I shall have to say will apply to diseases of other organs as well. But to determine what is syphilitic becomes of particular difficulty with regard to nervous diseases. Therefore it is in relation to them that the problem can be discussed and studied in its entirety. This question is by no means a purely academical one, as you may perhaps be inclined to think. It is not only of great scientific interest, but, as I hope to convince you, of far-reaching practical bearing. This, to mention only one instance, becomes apparent in weighing the chances which the syphilitic has with regard to life assurance.

For a long time syphilis of the nervous system has been considered the prototype of what is called *tertiary* syphilis. But the inadequacy of the time-sanctioned division of syphilitic lesions into

* Lecture delivered at the Medical Graduates' College and Polyclinic on March 9th, 1904.

Digitized by Google

secondaries and tertiaries is perhaps nowhere more apparent than when applied to nervous diseases. Originally the division was evidently made from a purely chronological point of view, and the difference between secondary and tertiary symptoms amounted to nothing more than a distinction between earlier and later manifestations of the disease. But *Ricord* already had, with this chronological principle, interwoven an anatomical, or rather a topographical, one. Affections of the skin, throat, pharynx, and nose, iritis, sarcocele, etc., were, according to him, counted secondaries; and affections of the bones, the muscles, the liver, the brain, etc., tertiaries. *Virchow*, justly exposing the weakness of this ambiguous classification, replaced Ricord's anatomical point by a pathological one. Not the seat of its lesion, but its nature became with him the decisive moment. The inflammatory processes became the secondaries, the gummatous the tertiaries. Such a division was a decided progress compared with its predecessor. But in application to clinical experience it also proved a Procrustean bed, into which the unwieldy form of the disease cannot be forced without considerable distortion of facts. For not only the inflammatory and gummatous changes are frequently combined, but also some of the latest affections of the nervous system may be purely inflammatory, while the gumma may appear at the very onset of the disease. I will not detain you by relating other attempts at classification which have been made in order to avoid the shortcomings of those just mentioned. None of them have found such a general acceptance as these two, and none have succeeded in putting the multiplicity and irregularity of the manifestations of this "most regular" disease upon a more satisfactory footing or basis. The principal defect of the usual classification is its hybrid nature—the intermixture of two, or even more, distinct principles of division. But, while aware of the unsatisfactory and inadequate character of this generally accepted classification, we are looking out in vain for something better. If we consider what we understand by the term tertiary, as distinct from secondary, we will find that by the latter we think of some early inflammatory infection of the skin or mucous membrane, while the former brings into one's mind some late gummatous affections of the bone or brain. In other words, we will find that the three different principles just mentioned, viz. the chronological, the anatomical, the pathological, are mixed up together. If the result is not satisfactory, the fault lies with the principle of classification, or, rather, with the unruly disease, which baffles all attempts at orderly arrangement. As I have said, this is most apparent where syphilis of the nervous system is in question. Of late our views with regard to the time of onset of nervous diseases in syphilis have undergone a complete change. Formerly brain and spinal cord were considered the parts last to be attacked by the syphilitic virus. Careful statistics, however, show that while the first year after infection furnishes a very large proportion of cases of nervous diseases, it is the first two years taken together which produce considerably more than half of all the nervous diseases to be observed in connection with syphilis.* Two, or even three, years is now the duration generally allotted to the secondary stage. It follows, therefore, that the majority of nervous affections in syphilis belong to the secondaries, or what is generally called so.

Some time ago I had to review a modern continental text-book of somewhat formidable dimensions. It was divided into the old classical parts: primary, secondary, and tertiary. In the volume treating of the secondary stage the functional disorders of the nervous system were alone dealt with, while the discussion of the organic diseases was entirely left to the tertiary period. This certainly is not up to date. For we now know that it is during the secondary stage that the nervous system is most liable to suffer. This fact will become still more evident if more discrimination is used before attributing a nervous lesion to syphilis, for no better reason than that it is either found in a person who has once suffered from syphilis, or that no other cause can be detected. Virchow has already given a timely warning not to throw everything into the "syphilitic swamp" for the simple reason that it is either found on a syphilitic, or that we do not know what to make out of it. The first is, of course, a grave and palpable fallacy, the second makes syphilis a kind of general receptacle for what must often remain doubtful in medicine. I remember many years ago having heard from the

* Vide 'Lancet,' June, 1895, "The Time of Onset of Syphilitic Diseases of the Nervous System, etc.," by George Ogilvie.

Digitized by Google

medical students in Vienna a little doggerel rhyme, to the effect that everything is syphilitic which the medico does not understand. It seems that even now-a-days a somewhat similar principle is not unfrequently applied in judging the part which syphilis plays in the causation of nervous disorders. To fallacies of this kind, the syphilographer is perhaps more liable than either the neurologist or the general physician. Syphilophobia has been defined as that state of mind in which a person sees syphilis everywhere. This mental condition —a kind of professional neurosis—seems to have given rise to some of the alarming estimates of the influence which syphilis exercises upon the nervous system.

It is generally granted that there is no nervous disorder characteristic of syphilis. Of course there are certain features—the multiplicity and transitoriness of the symptoms, the proneness to relapse, etc.—which will favour the diagnosis of syphilis. But even these, syphilis has in common with non-syphilitic nervous diseases, such as hysteria and multiple sclerosis. On the whole, there is not a single clinical criterion by which we can with any degree of certainty declare an affection of the nervous system to be syphilitic. Consequently our diagnosis rests on three points: (1) a previous history of the disease, or traces of past or present syphilis; (2) the possibility of excluding other causes; (3) the effect of antisyphilitic treatment. With regard to the *first* point there is the possibility of a double fallacy: that everything found in a syphilitic is not necessarily due to syphilis; secondly, that the absence of all evidence with regard to syphilis, past or present, does not in any way disprove the syphilitic origin of the nervous disease. The *second* point is of a still more intricate nature. With the unsatisfactory state of our insight into the ætiology of nervous disease in general, the inability to find some other plausible explanation for the existence of some nervous disorder amounts to a confession of the limitation of knowledge, rather than to the "exclusion" of other possible causes. Moreover, it is rare to find cases in which a careful and complete examination of the patient's general health, hereditary tendencies, habits, etc., fails to reveal some other condition which might have had a noxious influence upon the nervous system. And with regard to the *third* point, my belief is that the diagnostic value of the so-called specific drugs is considerably overrated; for not only are we constantly open to the old *post hoc, propter hoc* fallacy, but also many nervous diseases, whether syphilitic or not, may greatly improve, and even disappear without any treatment, specific or otherwise. And we must remember that the inefficacy of the treatment proves nothing against the syphilitic nature of the complaint, while at the same time it seems quite unlikely that the efficacy of such powerful drugs as mercury and iodide of potassium should be limited to one particular disease. Let me remind you of Mr. Hutchinson's warning that those who, for diagnostic purposes, rely upon the effect of "specific" drugs will make grave mistakes. With regard to mercury, it will be generally admitted that its curative effect extends beyond the domain of syphilis. To the preparations of iodine, on the other hand, an almost exclusive action is ascribed by many. Statements of this kind always invite a close scrutiny of the evidence upon which they rest. If we take the rough and ready routine treatment of organic disease of the nervous system of *doubtful* origin, it consists first and foremost in the application of antisyphilitic treatment. According to general usage, its efficacy is admitted as sufficient proof of the syphilitic nature of the disease. If, relying on this principle, we adduce such cases as proof of the specific effect of the drug, we are of course moving in a vicious circle, and I feel sure that many of the statements with regard to the so-called specific action of these drugs rests on such false reasoning.

Of course where all three points can be satisfactorily established, *i.e.* where there is clear evidence of present or past syphilis, no concurrence of other ætiological factors, and a speedy or more or less complete recovery under specific treatment, the diagnosis can *intra vitam* be made with a probability, practically amounting to a certainty. Even then mistakes are possible. But cases in which all these conditions are fulfilled are comparatively rare. If one goes carefully and critically through the profuse literature of the subject, closely examining the evidence given, one is struck by the levity with which the diagnosis of syphilis is frequently made. If there were such a thing as the Scotch verdict "not proven" in syphilography, syphilis of the nervous system would shrink sur-

Digitized by Google

prisingly. The statement, for instance, that tertiaries of the nervous system are more frequent than tertiaries of other organs is explainable by an individual experience, or perhaps by an indiscriminate and hasty procedure in diagnosing syphilis.

We must add that, even histologically, the diagnosis of syphilis of the nervous system cannot be made with absolute certainty from the anatomical appearance alone. We know that in syphilitic diseases of the nervous system the nerve substance remains primarily intact; that the changes which it undergoes are secondary to gummatous growths, meningeal exudations, and vascular alterations which disturb its nutrition. But these changes are, one and all, *not* pathognomonic. The gumma, the obliterating arteritis, once considered as truly specific, have, with the progress of histological research, lost this claim.

The discovery of the specific organism, which has so long been present to the mind's eye of the pathologist, will do much to advance our knowledge of the syphilitic process, and settle much which now must remain doubtful. However, no oversanguine hopes for the clinical differential diagnosis must be attached to its discovery. We know that the question, What is tuberculous? has by no means been fully settled by Koch's discovery. The presence of the bacillus is not proof of tuberculous disease, while its absence is no proof against the tuberculous origin of the anatomical lesion. Moreover, the *intra vitam* diagnosis of syphilis of the nervous system is not likely to gain materially by the detection of the parasite, and it is the clinical aspect of the question which alone is considered on this occasion.

From the foregoing it will appear that the diagnosis of syphilitic disease of the nervous system is to a large extent a matter of great difficulty and uncertainty. Syphilis is a mimicking disease, its manifestations are more or less imitations of other pathological types, it lacks originality. It is perhaps this want of specificity in its symptoms which, by a strange contradiction, but according to an old adage, has gained for it the designation of the specific disease *par excellence*. Of such contradictions many more are to be found in syphilography, and perhaps none are more striking than that syphilis has been, and still is, considered a "surgical malady." On this point, although it is not directly connected with my theme, I must ask your indulgence for a moment. Syphilography—in this country at least—has long been, and still is, considered a part of surgery, although there is scarcely any other disease in the whole range of medicine which is so intrinsically "internal," and so rarely appeals to the skill of the *chirurgeon*. Mr. Hutchinson once gave it as his opinion that, "had it not been for the unfortunate tendency to recur and become protracted, syphilis might have remained a surgical malady." Yet he himself describes it as one of the "specific fevers," and classes it with such diseases as variola, measles, scarlet fever, etc. It seems to me that, like the "specific fevers," or as we would say nowadays, the acute infectious diseases, syphilis never has been a surgical malady, and that the physician has some better-founded claim to it than the surgeon, who bountifully bestows on him all that has the unfortunate tendency "to recur and become protracted." I do not flatter myself that what I have said will have the effect of restoring syphilography to its proper place and quarters. But I wish to give expression to a long felt grievance arising out of a wrong division of labour.

I now return to the syphilitic diseases of the nervous system, which should be relegated to the physician, not only for their unfortunate tendency to be obstinate, but also for the difficulty and uncertainty of diagnosis. Here, more than anywhere else, it is true that the diagnosis of syphilis cannot be made from a single symptom, but only from a combination of symptoms, or rather from the evolution of the disease. A causal connection between syphilis and a nervous disorder cannot be established by the report of isolated cases, but only by the consideration of large numbers of cases of the same kind. This difficulty of diagnosis is further increased if we turn from the syphilitic diseases of the nervous system to those called parasyphilitic. The definition given of a para syphilitic affection is, that it is syphilitic in origin but not in nature. In other words, that it is caused by syphilis, but that it is neither inflammatory nor gummatous. In the case of nervous diseases the decision whether the disease is syphilitic or parasyphilitic would therefore rest entirely with the pathologist, were it not for certain clinical differences by which their nature is said to manifest itself *intra vitam*. Fournier, the foremost protagonist of parasyphilis, gives the two following rules by which

Digitized by Google

to discriminate between syphilitic and parasyphilitic affections. *Firstly*, the parasyphilitic lesion is "banal," it is not characteristic of syphilis, it is to be met with outside of syphilis, it is produced by other causes, while the syphilitic lesion, such as a mucous patch or a gumma, is "specific," *sui generis*, and is not to be found in any other disease but syphilis. *Secondly*, the effect of antisyphilitic treatment on parasyphilitic lesions is said to be either *nil* or very slow and incomplete, while in syphilitic affections it is rapid and complete.

At the last International Congress the value of parasyphilis, for the retrospective diagnosis of the disease, was one of the subjects under discussion. In my address there * I tried to show that the principle underlying the distinction between syphilitic and parasyphilitic affections is not only insufficient, but that it is faulty altogether. In its application to hereditary syphilis in particular it leads to consequences which by themselves prove its fallaciousness. But this becomes most evident when this principle of division is applied to syphilis of the nervous system. We have already seen that all truly syphilitic nervous diseases are "banal;" there is not one which is specific or characteristic of syphilis, not one which may not be produced by other causes as well. And we have also seen that the inefficacy of treatment constitutes no certain proof of the syphilitic nature of the disease. In fact, in syphilis of the nervous system the chances of treatment depend upon the damage done to the nerve substance by the alteration of the connective tissue, and where such damage has been of considerable extent or duration, the effect of treatment will be practically *nil*. It must always be remembered that in organic nervous diseases there is a difference between the anatomical and the clinical cure. Specific treatment may heal the syphilitic processes without any amelioration of symptoms, because the nerve-tissue has suffered beyond repair. In nuclear lesions of the third nerve, for instance, consequent upon syphilitic inflammation, it seems that treatment is rarely of any avail, owing to the tenderness and vulnerability of the nervous elements concerned. In all such cases the effect of treatment, clinically speaking, will be negative, while anatomically it may have reduced the process of cell-proliferation to cicatrisation. But not in these cases only is antisyphilitic treatment doomed to be powerless, although directed against a lesion truly syphilitic in nature. Let me remind you of those fortunately rare cases of acute transverse myelitis occurring in the early stage of syphilis. It has long been observed that these cases of sudden onset have a tendency to run a rapid course, and are rarely checked even by the most energetic treatment. During a marked secondary rash with more or less rise of temperature—and while under appropriate treatment—the patient develops suddenly all the classical and "banal" symptoms of a transverse myelitis and succumbs, although treatment is pushed to the utmost. The post-mortem reveals the diffuse changes in the arteries typical of syphilis. Cases of this class would have both the characteristics of parasyphilis, viz. banality and refractoriness to treatment, and yet be true syphilis. The greater or lesser completeness of the curative effect, its greater or lesser rapidity, are marks too vague and indistinct to build upon them a differentiation of two classes of affections. With regard to the last point it has been my experience that, unless some improvement is noticeable by the end of the *second week*, the chances of a cure, whether partial or complete, become very small. This, of course, is not a hard and fast rule, without exceptions, but with the beginning of the third week the chances certainly become rapidly less favourable. Incomplete cures are so frequent with true syphilitic diseases of the nervous system that with some of them, as for instance Erb's spastic spinal paralysis, they are the rule. On the other hand, more or less improvement and even complete cures by treatment have been reported in cases of parasyphilis, and if we are to accept certain statistics, some parasyphilitic lesions, such as tabes and leucoderma, are more frequently associated with a distinct history of syphilis than even the gumma itself. Of this I shall have to speak anon. It is the faulty clinical principle itself, upon which the whole distinction between syphilis and parasyphilis is based, that of necessity leads into a maze of contradictions. Between the two we are told there exists an "abyss," yet the same author declares that there is no clear line of demarcation between them, that one gradually and continually passes into the other.

Parasyphilis, if not the great "invention" which

* Vide 'Lancet,' June, 1903, "An Address on Parasyphilis, etc.," by George Ogilvie.

Digitized by Google

it has been proclaimed, is not altogether a bad nut. Besides a large bubble of air it contains a small substantial kernel. But this, on closer inspection, turns out to be an old friend dressed and disguised in a neologism. Pathological conditions which follow a certain morbid process, from which they derived their origin, without partaking of its nature, have long been known as the *sequelæ* of a disease, and have formed a difficult and curious field of investigation for both the clinician and the pathologist. Sir Dyce Duckworth's interesting and comprehensive lectures on the subject (1897) may still be fresh in the memory of some of you. It has also long been known that syphilis, like other infectious diseases, may lead to disturbances of a general or a local character, to degenerative processes. Lardaceous disease, frequently caused by syphilis, was by its discoverer declared to be "void of all specificity," to be syphilitic in origin, but not in nature. Cachexia, marasmus, pulmonary phthisis, have long been acknowledged as consequences of syphilitic infection without being part of it, *i.e.* notwithstanding their banality or want of specificity. By some authors the term "quarternary" stage has been coined for these non-syphilitic after-effects of syphilitic infection, while by others they have been called post-syphilitic, metasyphilitic, or parasyphilitic. Among these last the choice of the most appropriate term will rest with the learned Athenian. But the thing to be expressed is of great antiquity. In fact, it may be said of parasyphilis that what is certain of it is not new, while what is known of it is not certain. It is certain that there are pathological conditions, syphilitic in origin, but not in nature; but whether all the pathological conditions which have recently been huddled together under this collective name have anything in common, whether in particular they can ever be traced to syphilis as their source, must, to every careful and sober observer, be a matter of serious doubt. To Mr. Hutchinson we are indebted for a happy parallel, illustrating the relation in which the sequelæ of a disease stand to the disease itself. They follow it, he says, as war is followed by famine. The two things differ *toto cælo*, yet the one is the cause of the other. It will be known to you that Mr. Hutchinson looks upon the tertiary stage of syphilis as the sequelæ of the true infectious disease, which, according to him, is represented by the secondary stage. This position, however, seems to me untenable. Tertiaries most decidedly belong to the period of active hostilities, to the true specific manifestations of syphilis; they certainly are syphilitic both in origin and in nature. To continue the same metaphor, the tertiary residues may perhaps be compared to the occupation army, which after the termination of the actual warfare still keeps the hostile strongholds, and must be considered as part and parcel of the invading force. With regard to parasyphilitic conditions, the simile seems to apply with particular appropriateness. Of course at the present day these after-effects of syphilitic infection are generally explained by the action of the problematical toxins of the missing germ. Like all bacteriological theories of syphilis, this is only patchwork made from scraps of evidence, clinical or experimental, taken from other infectious diseases. Surely no one to-day will feel inclined to subscribe to the saying of one of the great masters, that "syphilis is the key to pathology." With better right it may be said that all the keys which pathology affords, have been tried in vain in order to "unbar these locks," behind which syphilis hides its true nature.

Two classes of nervous diseases have been described as parasyphilitic, viz. functional disorders and degenerative processes. Among the former we find neurasthenia, hysteria, and epilepsy; among the latter, tabes and general paralysis. Of certain other affections, such as hydrocephalus, I shall not speak here, because they belong to the congenital form of the disease, and in this lecture I am limiting myself to acquired syphilis. Nor shall I speak of the psychical disturbances, the true psychoses following syphilis. On this subject, only last year, Dr. Savage lectured to you—than whom we have no more competent authority. You know that there is no definite line of demarcation to be drawn between neurasthenia and hysteria. It is therefore practical to treat these sister-affections together. That syphilis may be among their causes—exciting, or occasional, there can be no doubt. But if you look through the long list of *agents provocateurs*, which in a neuropathic disposition may elicit the manifestations of these polymorphous neuroses, you will find nearly every other morbid state, whether chronic or acute, general or local. It would certainly not conduce to a clearer understanding of their nature, nor to a more convenient nomenclature

Digitized by Google

of nervous diseases, if exactly the same symptoms, or combination of symptoms, would in every case be called after their exciting cause with the prefix "para." That influenza, tuberculosis, even a simple cold, may in the neurotic be followed by nervous disturbances of various description, everyone knows. Yet nobody would speak of para-influenza, para-tuberculosis, etc., of the nervous system. That syphilis may act in the same way, "needs—as Horatio says—no ghost to come from the grave to tell us." But to apply the term para-syphilis to these nervous symptoms seems to me, to say the least, superfluous. It is true that in some cases they outlast the symptoms of syphilis proper, and show no tendency to disappear under treatment; in other cases, however, they vanish with, or even before, the truly syphilitic manifestations after a short application of specific drugs. Thus we should be forced to assume two forms of neurasthenia and hysteria, both caused by syphilis; one syphilitic, one parasyphilitic; one amenable to treatment, the other not. Fournier, in his new treatise, devotes a chapter to the functional disorders of the nervous system during the secondary stage, while he also treats exactly the same affections as specimens of parasyphilis in another work. In the former he recommends mercury as the sovereign, the only and certain remedy; in the other he denounces it to be utterly useless, or at least of very slow and incomplete effect. There is an "abyss," we are told, between syphilitic and parasyphilitic neurasthenia! I think you will agree with me that such a division is forced and artificial, purely theoretical, of neither practical nor scientific value. Syphilophobia, for instance, a not very unfrequent and often a most troublesome complication of the disease, as a rule outlasts all manifestations of syphilis, does not yield to treatment in the least; it may also exist without syphilitic infection; it has, in fact, all the characteristics of a para-affection. But exactly the same is true with what the French call *uréthromanie*, a morbid dread of *la goutte*, which occurs with or without gonorrhœa. To speak of it as para-gonorrhœa of the nervous system because it is "banal" and does not give way to astringents, would be preposterous. Yet functional parasyphilis of the nervous system does not rest on more solid ground. It is the *nature* of the disease which is said to establish the difference between syphilis and parasyphilis. But what is the nature of a functional disorder? I do not know of any better definition of a *functional disease*, than that it is one of which we do *not know the organic cause*. If, with regard to organic changes of the nervous system, the pathologist can frequently settle the question whether it is syphilitic or not, he must of necessity fail where he has to deal with functional disease. It is idle to speak of the "nature" of a disease, or to make the nature of disease the principle of classification, where the inscrutability of its nature is part of its essence.

That functional disorders may accompany and unpleasantly complicate syphilis, there is, as I have said, no doubt. But the frequency with which this occurs must, to a large extent, depend on national, or rather racial, peculiarities, if we compare the reports of French authors with what we see every day in this country. A distinguished French neurologist has expressed his opinion that hysteria in all its different forms is as common in England as in France, and that statements to the contrary are rather due to the unacquaintedness of English physicians with the picture of the disease than to a lesser liability of the English to suffer from it. Such a position is altogether untenable. I think it may be safely said that nowadays the picture of the disease, from slight neurasthenic symptoms to the alarming *ensemble de la grande hystérie*, is well known to every English physician, thanks to the remarkably vivid descriptions by French writers. But there can be no doubt that, in the male at least, hysteria is much more rarely observed with us than with our neighbours, according to their own reports. And as hysteria in general is less frequent with us, it is naturally a rare complication of syphilis. In men it is quite uncommon, but even in women it is not a frequent occurrence; and where it is to be found the causal connection between it and syphilis will frequently remain doubtful. I have for a long time given special attention to this point, and with particular interest sought for the objective symptoms of hysteria in the syphilitic. But the result is purely negative. Never have I found the concentric limitation of the field of vision—this classical feature of the hysterical picture—nor any one-sided anæsthesia. To find certain areas or patches of diminished sensibility in women is so frequent, that it is of no diagnostic value. After protracted medical examination it is always easy to map out such insular spots. But

Digitized by Google

they are artificial products, due to the overstrain of an unduly prolonged exploration.

Much has been said on analgesia during the secondary stage. But here again my personal experience is negative. Of course, if the over-zealous scientist gets the better of the considerate physician, and continues his pin-pricking or other experiments for a considerable period, he is pretty sure to find what he wants, partly from mental fatigue of the patient, partly even from exposure of the skin to the air. But if these sources of error are taken into due consideration, as they always should be, analgesia will—with us at least—be found to be a very rare associate of syphilis, and, where it exists, to have more the character of a complication or casual accompaniment than of a consequence or sequel. Tables have been drawn up to give with much minuteness the characteristic points by which syphilitic analgesia is to be discriminated from the ordinary, *i. e.* the non-syphilitic form. But here again the distinctions made seem arbitrary and fictitious. Whenever analgesia appears in a patient of hysterical disposition as a consequence of syphilitic infection, it is sure to differ in no way from that due to other causes, and not to betray its origin by petty peculiarities of localisation, intensity, or duration.

It is not only in frequency that the functional disorders consequent upon syphilis seem to differ according to racial disposition, but in quality as well. In fact it seems that certain manifestations of hysteria, not infrequent with the syphilitics of France, are never to be met with here. As an instance I want to give a phenomenon upon which Fournier lays particular stress, viz. boulimia. Perhaps those of you who have, in the toil of professional work, dropped some of your Greek will be in want of an explanation of the word. It is what we call a canine or dog appetite, or the hungry evil, a voracious hunger. This condition, as was to be expected of a nation of beef-eaters, is not unknown in this country. Nor are classical witnesses wanting to describe its occurrence among us. Sir Thomas Browne* has, under the heading "Boulimia Centenaria," reported a case of boulimia occurring in a woman 102 years old. He was "ready to afford his charity unto her," but "I should be loth," he adds, "to spend a piece of ambergris I have upon her, and to allow six grains to every dose till I found some effect in moderating her appetite." If rare in a centenarian, it is otherwise a fairly common complaint with us. That uncomfortable play "Little Mary," which has delighted many of you, is perhaps intended as a specific, a kind of ambergris against boulimia as a fashionable society disease. That to be "a great eater of beef does harm to the wit" was Sir Andrew Aguecheek's opinion, backed by personal experience. A reaction upon the higher functions of the nervous system may consequently be expected in a "beef-witted" race. Yet boulimia as a symptom of syphilis is, as far as I am aware, unknown in this country. Fournier describes even two forms of it, one syphilitic, belonging to the secondary stage, amenable to specific treatment and curable by such treatment only, the other parasyphilitic, refractory to specific treatment, or influenced by it only to small extent. Of the first form I feel confident no case has occurred here. A voracious appetite satisfied with mercury, would be considered too extraordinary an event to be overlooked by any medical man, even if he were not quite up to the latest subtleties in diagnosing hysteria. Whenever boulimia occurs here it is of the more expensive, of the "para," type, and in spite of ambergris, there is no specific for it like beef. But that it is ever caused by syphilis, even when it attacks a syphilitic, would be hard to prove. Although this symptom has no practical importance for us, its consideration is not without some use in another respect. It teaches a valuable lesson, viz. that every disease has to be studied separately in every individual country, that accounts of it emanating from the Continental schools cannot on faith, and without counter-proof, be accepted as also applying to our insular constitutions. If this is particularly true with regard to nervous diseases, and among them to functional disorders, it is also true in relation to syphilis and its influence upon the nervous system. Observers, as well as diseases, show racial peculiarities. A mind of high imaginative power will see with an eye different to that of a robust matter-of-fact intellect. As I have said before, syphilophobia has its victims among

* Mr. Wilkin, the learned editor of Browne's works, reminds us that Plutarch narrates that Brutus was attacked with this "disease" on his march to Durrachium. I have been told that it is no uncommon complaint with soldiers engaged in prolonged marches, especially if they lead through a poor country.

Digitized by Google

the workers on syphilis. Indeed, much of what is proclaimed as parasyphilis, particularly in the hereditary form of the disease, can be traced to a kind of syphilographic boulimia, a craving to incorporate every pathological condition, without discrimination, into the voracious viscera of one speciality.

That epilepsy is occasionally produced by syphilis seems to be generally accepted. Of course I am not speaking of cortical, symptomatic, or Jacksonian epilepsy; its connection with syphilis has long been established beyond doubt; both arterial changes or gummatous growths may lead to it. The knowledge of functional epilepsy as a consequence of syphilitic infection is of more recent date. The question here is of particular difficulty because most of those who accept syphilis as one of the causes of functional epilepsy yet declare specific treatment to be powerless. Fournier's declarations on this point are also contradictory, as he describes two distinct forms, one which he calls secondary epilepsy, easily influenced by mercury, and a parasyphilitic form upon which the drug has no effect. That in some cases a syphilitic infection is the only possible cause in the patient's previous history, which may be held responsible for the outbreak of his epilepsy, is an argument of doubtful weight, because in functional diseases in general, and in epilepsy, in particular, the most minute search is frequently unable to discover any ætiological factor. On the other hand, it is rarely possible to eliminate completely all other causes of epilepsy, such as heredity, trauma, mental shock, alcoholism, acute infectious diseases, heart disease, arteriosclerosis, etc. The neuropathic disposition in particular is so indefinite an entity that to exclude it is practically a matter of impossibility. In the ætiology of functional disease, and in epilepsy particularly, we have frequently to content ourselves with a Socratean confession of ignorance. It is the "shrewd philosopher" who has a wherefore for every why, not the seeker after truth. In favour of syphilis as a cause of epilepsy will be its appearance after the twentieth year, or between that time and old age. The first appearance of ordinary epilepsy after forty is not as rare as has been formerly believed, while in the great majority of cases it makes its *début* during the first two decades of life. That every case of epilepsy which is met with after a certain age, say after thirty, is syphilitic is an unwarranted exaggeration. Where there is no history of syphilitic infection, the onset of epilepsy after thirty years of age is still to be observed in from 5 to 7 per cent. of the total. My personal experience of syphilis as a cause of epilepsy is small and inconclusive. It is easy enough to produce cases of syphilitic epileptics, but those with any claim to conclusiveness with regard to the causal connection of the two affections are rare. I do not recollect having seen more than two cases in which I felt justified in attributing the epileptic state to probable syphilitic infection. In one of them a sister of the patient had committed suicide; but the fits, with biting of the tongue and enuresis, appeared first at the age of thirty-three, three years after infection, and led within six years to considerable mental decay. It has been stated that syphilitic epilepsy does not in the course of time bring on that deterioration of the general mental faculties which as a rule follows the genuine form. But here again it has to be remembered that there are a good many epileptics who, after long periods of close observation, present no appreciable intellectual defects, while some authors have reported considerable degrees of dementia in cases of syphilitic origin. In my cases specific treatment was apparently of no avail. But to judge of the effect of treatment in epilepsy is a matter of great uncertainty, because there are many cases of epilepsy without a previous history of syphilitic infection, which without treatment show long-lasting remissions. That syphilis is among the causes of true epilepsy is a statement which I have to accept on authority, as I am not able to support it from my own observation. The "nature" of this epilepsy, its pathological substratum, is, like that of epilepsy, altogether a matter of mere conjecture. The so-called secondary epilepsy may be due to minute specific alterations in the grey matter of the brain; the parasyphilitic form is by some ascribed to the action of toxins. But little is gained in "explaining" the unproved by the unprovable, *ignotum per ignotius*.

Whether tabes and general paralysis are only two forms of the same process differing by the localisation of the morbid changes, or two distinct pathological conditions, is a question still *sub judice*. It is not my task to describe to you the

Digitized by Google

clinical or anatomical picture of these diseases, but only to discuss the reasons for which they are generally considered as after-effects of syphilis, and how far this assumption is justified. For this purpose they may conveniently be treated together. It is therefore sufficient here to consider the connection between tabes and syphilis. The views expressed in this respect will, with slight modifications, also hold good for general paralysis.

A few introductory remarks on the pathological process underlying the clinical picture of tabes are necessary for a full understanding of the question under discussion. Tabes is one of the system-diseases of the spinal cord—a primary degeneration of the posterior columns. But we know through recent investigations that the classical picture of tabes, which undoubtedly is familiar to all of you, may in rare cases be produced by a truly specific meningitis compressing the posterior roots, to which the degeneration of the posterior column is only secondary. Syphilis, as I said before, is intrinsically a mimicking disease. These cases are fitly excluded from the range of true tabes under the name of syphilitic pseudo-tabes. We also know that true tabes may complicate itself with more or less extensive syphilitic inflammation of the blood-vessels of the spinal cord. To one or the other of these two classes belong, in all probability, all those cases in which specific treatment is reported to have had a favourable effect. We further know that, as in every other chronic degenerative affection of the spinal cord, chronic alterations of the arterial walls may be found in the cord of the tabetic; but these changes do not represent the primary lesion, and are in no way characteristic of tabes. In an individual case it may, therefore, be difficult to determine with certainty the starting of the morbid process. The view, however, that the degeneration of the posterior columns is always secondary to vascular disease, and that the latter is of syphilitic origin, *i. e.* that tabes is only one of the examples of spinal syphilis, has been rejected by the most competent workers on the subject. By a general consensus of opinion, in which the adherents of the syphilitic origin of tabes join with its opponents, tabes is regarded as a primary degenerative process in the nerve-substance, entirely distinct from genuine specific disease of the spinal cord. Quite recently the opinion has been advanced by a neurologist of note that in all cases of tabes the primary lesion is a true syphilitic inflammation of the posterior lymph-vessels of the cord; but up to the present this statement stands without confirmation. Consequently we must accept the primary non-specific nature of the tabetic process as an established fact.

Syphilitic, not in nature, but in origin—this was the definition given of a parasyphilitic lesion. Now let us consider the facts upon which the syphilitic origin of this non-syphilitic disease is assumed. The most important arguments for it are *certain statistics*, which prove the existence of previous syphilitic infection in patients suffering from tabes; but before submitting these statistics to a closer analysis I shall briefly mention the other facts pointing in the same direction. It has long been observed that both tabes and syphilis of the central nervous system affect with predilection certain nerves or groups of nerves, in particular those supplying the ocular muscles. These ocular palsies are frequently transitory in character; in both the optic nerve is prone to be affected, and the pupillary and patellar reflexes are influenced in the same manner. A number of cases have been reported in which syphilis acquired late in life was followed by an uncommonly late appearance of tabes. Tabes has been found in both husband and wife; in such cases there is always a clear history of syphilis. In other instances one of the two suffers from tabes, the other from general paralysis. Here, too, previous infection can be proved in at least one of the patients. Inoculation experiments with fresh syphilitic virus on persons suffering from general paralysis have given negative results; similar experiments on the tabetic are not known. Although in most cases of true tabes there is no corroborative evidence arising from syphilitic affections of other organs, there exists now a series of cases in which such co-existence has been observed. In particular, in cases of infantile tabes and general paralysis there is frequently certain evidence of congenital syphilis. In the absence of post-mortem examination, however, it must remain doubtful whether these cases do not belong to the category of pseudo-tabes. At a comparatively early age tabes has been observed, together with aneurysm of the aorta, apparently due to arterio-sclerotic changes. Childlessness is frequent in married women suffering from tabes, and may be

Digitized by Google

either due to want of conception, to abortion, or to the early death of the child—usually occurring during the first month. I cannot here discuss at full length the relationship between each of these facts and the question under consideration. If taken singly, some of them may be open to a different construction; yet, taken together in their entirety, they undoubtedly strongly support the causal connection between syphilis and tabes. The most important argument in favour of such connection is, as I have said, derived from certain statistical inquiries. Here, however, the results vary between the widest limits. While some have found not more than from 15 to 20 per cent. of syphilitics among the tabetic, others report 90 per cent. and more. It is impossible to estimate accurately the spread of syphilis among a population. But, from certain statistics collected in hospitals and private practice, it seems that in patients suffering from other nervous diseases than tabes or general paralysis, or from other diseases whether nervous or not, a history of syphilis may be traced in about 20 per cent. Perhaps Fournier's estimate for Paris, that between 15 and 20 per cent. of the population are syphilitic, comes near the truth also with regard to other large cities. If these calculations are only approximately correct, it will appear that, according to some observers, the percentage of syphilis in tabes is not greater than what corresponds to the general distribution of that disease; while others report exorbitant figures, even approaching 100 per cent. According to the former, syphilis would have no part in the production of tabes; according to the latter, it would be its predominant, if not its only, cause. Moebius, for instance, and Marie consider syphilis as the exclusive cause of tabes. According to them, every case without exception is of syphilitic origin. Indeed, if the ninety-and-more-per-cent. statistics were reliable, this seems to me the only possible and unavoidable conclusion, for the simple reason—as far as I know not yet pointed out—that these numbers are far in excess of those which are obtainable in cases of undoubted tertiary syphilis. For this very reason I believe that these excessive statements are not trustworthy. Neisser, the discoverer of the gonococcus, has for many years carefully collected the evidence of previous infection in cases of undoubted tertiary syphilis, in order to study the effect of specific treatment in the prevention of tertiaries. His name is known to all of you, and he certainly cannot be suspected of want of skill in detecting the *vestigia veteris flammæ*. Only such cases were entered in his statistics in which the diagnosis of tertiary syphilis was "absolutely certain," and in which there were reliable dates regarding previous treatment. He consequently excluded cerebral and visceral cases of doubtful origin—parasyphilitic affections like tabes, general paralysis, leukoplakia, also malignant syphilis. Yet the percentage of cases in which the infection was "unknown," in which the tertiary lesion was the *first appreciable manifestation* of the disease, varied from between 30 and 50 per cent. Fournier, among his cases of gumma o the tongue, found more than 40 per cent. without evidence of previous infection. Similar experiences have been reported by other observers. These numbers seem to me to be in flat contradiction to those reported in cases of tabes, unless tabes is a disease *plus syphilitique que la syphilis*. A close consideration of some of these extraordinary statistics seems to confirm the opinion thus formed of their validity.

Two years ago Fournier gave a summary of a thousand cases of tabes. The cases were "chosen from among many others." He made it a point to include only "precise, incontestable facts, relying on information intelligently given by the patient, on well-established antecedents, sometimes even on actual manifestations of syphilis." "In one word, a thousand cases where either the existence or the absence of syphilis in the patient can be affirmed categorically." This eclectic character of his series seems to deprive it of most, if not of all, demonstrative power. In the first place, to exclude categorically syphilis is an utter impossibility, particularly if one believes, like Fournier, that congenital syphilis may, with or without previous symptoms, make its appearance up to the age of sixty. But in order to determine the percentage of syphilitic infection it is, of course, indispensable to include all cases of tabes. The large number of cases, the "many others" in which previous infection remains doubtful or could not be proven, although it could not be "excluded," form as important a part of the statistical material as those either strictly negative or positive. This omission is the fundamental fault of Fournier's calculation.

Digitized by Google

Once more syphilographers' statistics are bound to be at variance with, and less conclusive than, those of the neurologist, because only those with a history or a suspicion of syphilis will, as a rule, seek his advice, while the others will remain with the neurologist or general physician. The result of Fournier's eclecticism is that he finds 92 or 93 per cent. of certain syphilis to 7 or 8 per cent. without antecedents, and he adds that this "quotient is a minimum only," because in from 6 to 10 per cent. syphilitic antecedents are missing in any syphilitic affection whatsoever. The contradiction in which he thus entangles himself is obvious, and is further increased by the emphatic statement that syphilis is *not the only cause of tabes*. If these 8 per cent. (the average between 6 and 10) have to be added in every syphilitic affection whatsoever, then we have for tabes a hundred brimful, and there is no other cause of tabes possible but syphilis; but then it also follows that in his 8 per cent. without syphilitic antecedents, the absence of syphilis could not be "categorically affirmed." Although I am not a "great arithmetician" like Cassio, yet I make bold to maintain that these more-than-ninety-per-cent. statistics are fictitious and fantastical figures.

I want briefly to refer to another recent statistical inquiry relating to the same point which also seems to me open to some criticism. Last year Dr. Byrom Bramwell published in "Brain" an interesting and instructive analysis of 155 cases of tabes. On the point in question, viz. the connection between syphilis and tabes, I cannot approve of the course adopted by him. He does not attempt to discriminate between hard and soft sores, and consequently takes a venereal sore with or without secondary symptoms as evidence of syphilis. This must materially impair the validity of his conclusions. Not only the soft sore, but, for instance, herpes of the prepuce may also be included in his statistics. He suggests that the toxins of the *soft* sore may produce the same effect on the nervous system, but up to the present we know nothing of the constitutional effects of the *soft* sore, much less of its toxins. In all probability the *soft* sore is not a pathological unity, but may be produced by several micro-organisms. Dualism, which thirty years ago Mr. Hutchinson pronounced to be "dead if not yet buried," seems to have revived and regained its old position.

If, for the reasons given, the excessive percentages arrived at by some observers are open to doubt, there yet remain other statistics which seem more worthy of credence, and would testify in favour of a close causal connection between tabes and syphilis. Sir William Gowers, for instance, found 50 per cent., and others have come to similar results. Even these numbers would, in the light of what I have said above regarding the evidence of past syphilis in tertiaries, go far to establish syphilis as a potent, if not the only, factor in the production of tabes. Indeed, taking this numerical evidence in connection with the other arguments briefly referred to, there seems to be little doubt about the syphilitic origin of tabes, a conviction impressed upon me by personal experience. Yet there remain certain apparently well-authenticated facts which seem to be in opposition to this doctrine. Among them, the most important seems to be that in districts and among tribes where syphilis has for long been raging endemically, *tabes and general paralysis are practically unknown.* The observations made by Neftel, Trenmen, Jeanselme, Düring, Glück, and others in different parts of the world agree that a population, although thoroughly impregnated with the syphilitic poison, may yet present *no specimens* of these "parasyphilitic" affections. Of general paralysis it has been said that for its development the co-operation of "syphilisation and civilization" is necessary. This *bon mot* would hardly apply to tabes, because the seat of civilization is not situated in the posterior columns of the cord. The rarity also with which prostitutes present tabetic degeneration is difficult to reconcile with its syphilitic origin. Yet, on the whole, it must be said that the evidence in favour of a close causal connection between tabes and syphilis is exceedingly strong, although some facts remain which seem to point to the conclusion that syphilis is not the sole cause.

Gentlemen, this is, as I see it, the present state of our knowledge as regards the causal connection between syphilis and tabes. There are still many points of interest and importance which I have only slightly touched upon, and which seem to invite a closer and more detailed consideration than could be given to them in the time at my disposal. Some of these may fitly engage our attention on a future occasion.

Digitized by Google

ON THE TREATMENT OF APPENDICITIS,*

By W. H. B. BROOK, M.D.Lond., F.R.C.S.,
Surgeon to the Lincoln County Hospital.

GENTLEMEN,—I have selected the treatment of appendicitis for our discussion to-night not only by reason of its very great importance from a medical as well as from the surgical point of view, but also because it is a subject which is as yet unsettled, upon which many diverse views are held, and for this very reason I have preferred to put before you a few of the more important points for discussion in place of reading a set and formal paper, in the hope that from the experience of all present we may be able to obtain some guidance for the future.

Appendicitis has come much to the front during recent years, so much so that all our better-class patients are familiar with it, and are able to talk about it, often sensibly and to the point; but if we look into any of our older text-books, *e.g.* the first edition of Fagge's 'Medicine,' published in 1886, we shall find no mention of "appendicitis;" although what is no doubt the same disease is described under the name of "perityphlitis," and is looked upon as having its origin generally in the cæcum, although a note is made that Dr. Wilks had ascertained post mortem that some cases were associated with ulceration or perforation of the vermiform appendix.

Our knowledge, therefore, of the cause and pathology of the malady has become much clearer and more precise during the past eighteen years, but it will be a matter for us to discuss whether our treatment has in every respect equally advanced with the times.

For the purpose of systematising our discussion I shall define appendicitis as peritonitis originating in the vermiform appendix as distinct from simple appendicular colic without peritonitis, but these two affections are so nearly related that we shall have to consider both in the matter of diagnosis and treatment.

Acute appendicitis may be divided into—

1. *Catarrhal appendicitis*—the mildest form,—which has a strong tendency to recover, but it is to be noted that in this catarrhal form the epithelium of the appendix, which is a single layer, is shed, and there is therefore a possible door open for an infection which may at any time spread to the deeper tissues, and we must also not lose sight of the fact that the bacteria—the *B. coli communis*—which are always present in the large intestine, and are, under ordinary circumstances, not only harmless, but of value in the final processes of digestion, have the power of becoming intensely virulent in the presence of unhealthy surroundings, such as are afforded them in a case of simple catarrhal appendicitis.

2. *Ulcerative appendicitis*, in which not only is the epithelium shed, but the mucous and submucous coats are opened up. The mischief may stop here if the deeper tissues are of good vitality and the microbes are destroyed, but, all the same, if the patient recovers he will carry about with him a damaged appendix with a weakened mucous membrane, and possibly a strictured lumen, leading to the formation of a cystic dilatation which contains mucus ready at any time to form a culture medium for the bacilli.

3. *Appendicitis (with or without perforation) and abscess.*—Here we may have—

(*a*) An abscess pointing through the abdominal wall;

(*b*) An abscess *in* the peritoneal sac more or less shut in by adhesions;

(*c*) An abscess *outside* the peritoneal sac, generally well to the right side, creeping up behind the ascending colon;

(*d*) Several localised collections of pus;

(*e*) General suppurative peritonitis.

4. *Appendicitis with localised adhesive peritonitis.*—This often ends in recovery, but recurrence is apt to take place.

5. *Fulminating appendicitis.*—The infection is of intense virulence, and the disease runs a rapid course, the patient dying from toxæmia in forty-eight to seventy-two hours, if not relieved by operation, and even then the mortality is from 75 to 95 per cent.

Symptoms.—The important symptoms in an attack of simple acute appendicitis are pain, tenderness, and rigidity.

The pain often follows an indiscretion in diet; it may at first be referred to the epigastrium or umbilicus, but after a little time—twelve to twenty-

* The introduction to a discussion at the Lincoln Medical Society.

Digitized by Google

four hours—is usually referred to the right iliac fossa, although I have seen a case in which the pain was referred to the left iliac fossa. It is sometimes difficult to distinguish this pain from pain in the right ovary, more particularly as an attack of appendicular colic may precede a menstrual period, probably caused by the ovary receiving part of its blood-supply from a branch of the artery to the appendix; the premenstrual congestion of the ovary would then be accompanied by a congestion of the appendix. Conversely a diseased condition of the appendix may cause ovarian pain and menorrhagia, and I can recall one case of my own in which removal of the appendix was followed by a cessation of the menorrhagia. The pain often radiates in the direction in which the appendix lies; thus it may be very marked at the umbilicus, over the bladder, at the internal abdominal ring, or in the lumbar region—an important indication if an operation is to be performed.

The tenderness, a most characteristic feature, generally most marked at McBurney's point, where a line drawn from the umbilicus to the right anterior superior iliac spine crosses the outer edge of the rectus; but, as Mr. Sherren has lately shown, it may be met with over any part of the area supplied by the eleventh dorsal nerve. This tenderness does not require deep pressure to elicit it; it may be brought out by simply pinching up the skin.

The rigidity is confined in a simple case to the lower right quadrant of the abdomen.

To these chief symptoms must be added *obstinate constipation; vomiting*, which usually subsides after the first few hours, except in the fulminating form of appendicitis; *fever*, except in a case of simple appendicular colic, or in a case of fulminating appendicitis which is rapidly going the wrong way.

The pulse is generally somewhat increased in frequency; its rate and character afford an indication of the progress of the case of the utmost importance, a steadily increasing frequency being the most marked sign of ill omen, whilst a steadily diminishing rate affords a good prognosis.

Such an attack may terminate in (1) complete recovery, the temperature falling to normal about the third day, the pulse steadying down, the pain subsiding, and the bowels acting; (2) localised suppuration; (3) apparent recovery, but with a tendency to recurrence; (4) a sudden change for the worse—fulmination.

Fulminating appendicitis is a violent explosion, which may be the first symptom of the disease, or may occur at any time during a simple attack, whether it be the first or recurrent.

The symptoms may be the same as those of an ordinary acute attack, but of greater intensity, the pain much greater, the tenderness all over the abdomen, with early tympanites. The pulse rapidly increases in frequency, becoming small and feeble; the face has an anxious "drawn" expression, and the skin becomes clammy. With the increasing frequency of the pulse the temperature often falls to below normal. The progress is rapid, the patient generally dying within forty-eight hours.

Having thus cleared the ground by a short consideration of the varieties and symptoms of appendicitis, we can the more readily discuss the treatment.

I. There is no question that in the case of patients who have had several attacks of simple appendicitis the correct line of treatment is to remove the appendix during a quiet interval. In careful hands this has a mortality of only about ½ per cent.

How many attacks should a patient, however, be allowed to go through before having the appendix removed?

Provided that a patient takes warning from his first attack and carefully avoids everything that might lead to another, such as eating indigestible substances, hurrying over meals, not masticating the food carefully, neglecting the regular daily action of the bowels, careless exposure to wet and cold, he may never have a second attack; but yet statistics show that from 25 to 44 per cent. of first cases relapse, the recurrence generally taking place within two years.

Such a recurrence indicates some permanent damage to the appendix, and a consequent liability to further attacks, any one of which may possibly fulminate.

I think, therefore, that removal of the appendix during a quiet interval should be strongly urged upon a patient who has had two attacks, although he would have been well advised to have the operation done after the first attack if it had been severe.

Digitized by Google

In respect to this I feel that the Workmen's Compensation Act has furnished a strong inducement for the intermediate operation after the first attack in all workmen employed in laborious work. Such an one is extremely liable to have a second attack precipitated by the stress and strain of his ordinary work, and his employers might be held liable for compensation. One could not therefore blame any employer who refused to take back any workman after an attack of appendicitis until he had been made safe by an appendicectomy.

II. *The treatment of a fulminating attack.*—Here the patient is in a desperate condition, which has been well compared to a strangulated hernia or a perforated gastric ulcer, and if left he will die within forty-eight hours, so that in such a case operation affords the only chance, and that only a slender one unless performed within the first twelve hours. Various statistics place the mortality attending such an operation at 75 to 95 per cent., whereas not 1 per cent. recover if left.

Fortunately only 1 per cent. of all cases (if we include the mildest) fulminate, but it is of the utmost importance to watch all for the onset of untoward symptoms, the chief being the steadily increasing frequency of pulse and the appearance of the patient.

III. *The treatment of appendicitis with suppuration.*—Sir F. Treves has laid down the rule that "immediate operation is demanded in every example in which there is reasonable suspicion that suppuration has taken place" ('Brit. Med. Journ.,' June 28th, 1902), but this is a matter upon which there is a great diversity of opinion, many surgeons preferring to wait until after the fifth day if there are signs that the pus is localised and shut in by adhesive peritonitis. Here we can only be guided by symptoms; if there is a large indurated mass to be felt in the right iliac fossa, and if the pulse is not increasing in frequency nor the temperature becoming higher, it is safest to wait until the pus has been securely shut off from the general peritoneal cavity, when it should be evacuated by an incision, care being taken not to disturb the adhesions or open the general peritoneal cavity. Statistics show that such cases do best if they can be tided over the fifth day before operating.

If, however, the patient's condition is becoming worse, the pulse increasing in frequency, and the tender area extending, then immediate operation is called for, the pus being freely evacuated and large drain-tubes put in.

In one such case that I saw successfully operated upon pus poured out from an incision below the umbilicus and in the right and left iliac regions. Four large drain-tubes were put in, down to Douglas's pouch in each iliac region and running up to the liver, and the patient steadily and speedily recovered.

Our hope of saving such cases has been immensely increased by the method of continuous subcutaneous infusion, many pints of saline solution being run into the thighs by Southey's cannulas and a simple syphon. (I saw an instance of this with Dr. Ellis, of Metheringham, which recovered.) But as a general rule, if the pus is localised we should wait till after the fifth day before operating.

(In hospital work and in some easily accessible private cases the frequent counting of the leucocytes in the blood is of great value in showing the commencement of suppuration, but the difficulty of carrying it out in the majority of cases will, I think, prevent its general adoption.)

IV. *The treatment of simple appendicitis during the attack.*—Here we are on extremely debatable ground, some surgeons urging early operation and removal of the appendix, others holding that we should wait and, unless the symptoms point to fulmination, endeavour to tide the patient over the attack by rest and medicinal treatment. It is with the hope of adding to our knowledge of such cases that I have brought this subject before you to-night. As Sir F. Treves (loc. cit.) says, "Hospital statistics are satisfactory only up to a certain point, since they of necessity deal with cases of the most severe type; the best record which could be obtained would be based upon the experience of a large number of men in private practice." We shall, I trust, be able to-night to add something of value to these records.

My own small experience is that such cases can with care be tided through the attack, and I cannot recall a single instance in which this has not been the case.

The line of treatment that appears to be most useful is—

(*a*) Absolute rest and quiet, the knees being placed over a pillow.

(*b*) The application at frequent intervals of hot fomentations to the abdomen, a piece of lint

Digitized by Google

thickly covered with the glycerine of belladonna being applied to the seat of pain under the fomentations.

(*c*) Very low diet, the food being the simplest liquid food. Milk, if given, should be diluted and peptonised.

(*d*) No attempt should be made to move the bowels until the fourth or fifth day, and not until the seventh if an abscess is forming, when calomel and a saline aperient, such as phosphate of soda, may be given, followed by an enema.

(*e*) A simple saline mixture, with hyoscyamus or belladonna, or with salicylate of soda if there is any suspicion of a rheumatic tendency.

(*f*) The flatulence is best kept in check by salol in powder.

Here two points present themselves for discussion—the advisability of giving or withholding opium, and the use of aperients.

With respect to opium, from time immemorial this has been the great sheet-anchor for physicians in all cases of peritonitis, relieving the pain and quieting the restless patient, but of late years there has been on the part of surgeons a strong revulsion against its use, with undoubtedly considerable reason, for not only does it paralyse the intestines and lead to tympanitis, but, by temporarily improving the pulse and general condition and by relieving the pain, it gives a fallacious appearance of improvement, masking the real condition of affairs often until it is too late.

My own inclination is if possible to do without opium, using hyoscyamus or belladonna as a sedative, but at the same time I think we are justified in giving hypodermically a quarter of a grain of morphia on first seeing the patient, if he is in pain and distress, and provided that the case is not one that requires almost hourly watching to determine the course which it is going to take. By giving the one dose of morphia we shall quiet the restlessness, thus putting the patient in the most favourable position for recovery, whilst by only giving a single dose the effect will have passed off by the morning, and there will be no fear of the symptoms being masked.

I do not advocate giving opium in cases that are about to undergo operation, except it be in the instance of a patient having to take a long journey to the hospital, perhaps having to travel fifteen to twenty miles by road. In such a case morphia might well be given, the hospital house surgeon being, however, informed of the fact.

As to the use of aperients, a simple saline aperient may be useful in a case of appendicular *colic*, to clear away any peccant substance, but we have to carefully discriminate between such simple cases and those which are more severe, and in which an aperient would at once disturb the inflamed parts, and probably lead to general peritonitis.

In true appendicitis the bowels should be kept quiet until after the fifth day, when a dose of calomel may be given, followed by a saline, of which a very useful form is the phosphate of soda in drachm doses every four hours. Its action may be aided by an enema, but with caution, bearing in mind the sometimes close relation of an inflamed appendix to the rectum. In this connection may I mention the importance of a rectal examination in many cases as affording information of the presence of pus and the extent of inflammation.

When general suppurative peritonitis has occurred the only chance of recovery lies in drainage and large continuous saline infusion, and it is then absolutely necessary to get the bowels to act, calomel then being given in one-grain doses repeated hourly. My friend Mr. Barnard informs me that in some such cases under his care at the London Hospital 40 to 50 grains of calomel were required, and in one case as much as 72 grains. The patient recovered.

In conclusion, I would recapitulate our position in dealing with appendicitis as follows:

1. The very great majority of cases will get over the attack if treated on simple medicinal lines.
2. During the quiet period the appendix should be removed so as to obviate another attack.
3. During an attack the patient should be carefully and frequently watched, and on any sign of fulmination operation should at once be done.
4. If there are signs of suppuration an attempt should be made to tide the patient over the fifth day, and then the pus should be evacuated, if possible, without opening the general peritoneal cavity. If the appendix presents in the wound it should be removed, but no elaborate search should be made for it. It may be removed later on.
5. In general suppurative peritonitis the use of drainage, massive infusions, and calomel.

I have to acknowledge my indebtedness to Mr. James Berry, Surgeon to the Royal Free Hospital, formerly Surgical Registrar at St. Bartholomew's, for valuable statistics of several hundred cases occurring at those hospitals.

Digitized by Google

INDEX TO VOLUME XXIII.

PAGE

Abdominal aorta, aneurysm of, by J. H. Bryant 71, 89
„ pain, diagnosis of, by C. A. Morton 232
Abscess, psoas, case of, by A. M. Sheild . 68
„ subpectoral, case of, by A. M. Sheild 67
Adenoids, cases that look like, by S. Paget . 110
Adrenalin, use of, in anæsthetic injections . 176
Albuminuria, by P. H. Pye-Smith . . . 129
Alkalinity of urine, to reduce . . . 136
Anæmias, intermediate, by E. Cautley . . 380
„ primary and secondary, by E. Cautley 362
Aneurysm of abdominal aorta, by J. H. Bryant 71, 89
Appendicectomy, discussion on . . 267, 282
Appendicitis, case of, by A. M. Sheild . . 68
„ treatment of, by W. H. B. Brook 413
Arthritis, varieties of, by Sir Dyce Duckworth 113
Asthma and the nose, connection between, by A. Francis 222
Aural cases, by S. Paget 107

Balneary therapeutics, by L. Williams . . 249
Barnard, Harold L., clinical demonstration . 188
Bennett, Sir William, on some points relating to rigidity of the spine 49
Bishop, E. Stanmore, proliferating papillomatous ovarian cysts 329
Blackwater fever, large mononuclear leucocytosis in, by F. Bushnell 59
Blood, normal and abnormal conditions of, by E. Cautley . . . 343, 362, 380, 395
Bowlby, Anthony, pain: its importance in diagnosis and its tendency to mislead . 289
Bradford, J. R., value of symptoms in diagnosis 193
Bramwell, Byrom, demonstration of cases 237, 254
Breast, case of cyst of, by A. M. Sheild . . 69
„ predisposing and pre-cancerous conditions of, by H. T. Butlin . . 337

PAGE

Bronchitis and emphysema, by F. J. Smith . 43
Bronchiectasis, by S. H. Habershon . 161, 183
Brook, W. H. B., treatment of appendicitis . 413
Bryant, J. H., on aneurysm of abdominal aorta 71, 89
Bursitis, case of gummatous, by H. L. Barnard 188
Bushnell, Frank, large mononuclear leucocytosis in malaria, blackwater fever, and lymphadenoma 59
Butlin, H. T., on difficulty in swallowing . 1
„ on predisposing and pre-cancerous conditions of the breast . 337

Cancer in boy of eleven 236
„ of tongue, case of, by D'Arcy Power . 15
„ Röntgen rays in treatment of . . 267
„ uterine, X-ray treatment of . . 296
Cancerous (pre-) conditions of breast, by H. T. Butlin 337
Cataract, spontaneous absorption of . . 304
Cautley, Edmund, on normal and abnormal conditions of the blood . 343, 362, 380, 395
Cerebral cortex, case of injury to, by H. C. Thomson 392
Chapman, Charles W., at the National Hospital for Diseases of the Heart . . . 349
Cheyne, W. Watson, thoroughness in surgery 33
Children, common symptoms of disease in, by R. Hutchison 353
Clarke, J. Jackson, on piles, their nature and treatment 177, 206
Collins, E. Treacher, on injuries and diseases of the orbit 241
Constipation, chronic, on the operative treatment of the conditions of the large bowel which result from, by W. A. Lane . . 209
Cough and spit, chronic, case of, by B. Bramwell 237
„ in children, by R. Hutchison . . 355

Digitized by Google

PAGE

Croupy respiration in children, by R. Hutchison 361
Cyst of breast, case of, by A. M. Sheild . 69
Cysts, ovarian, by E. S. Bishop . . . 329

Doran, Alban, on broad ligament tumours . 149
Duckworth, Sir Dyce, on varieties of arthritis 113
Dupuytren's contracture, injections of thiosinamin in 144

Eclampsia, treatment of . . . 88, 240
Eczema, is it symptomatic? by T. Robinson 111
Emphysema and bronchitis, by F. J. Smith . 43
Enlargement of heart, by W. H. White . . 17
Epididymitis, case of gonorrhœal, by D'Arcy Power 16
Epilepsy and its treatment, by J. Taylor . 26
„ relation to gastro-intestinal disease . 299

Fasting in treatment 362
Fenwick, W. Soltau, hyperæsthesia of the stomach 296
Fox, T. Colcott, on impetigo 214
Fracture of ribs, case of, by A. M. Sheild . 65
„ skull, by T. P. Legg . . 153, 166
Fractured humerus, case of, by A. M. Sheild . 67
Francis, Alexander, on the nature of the connection between asthma and the nose . 222

Gastric surgery, by D'Arcy Power . . . 97
„ ulcer, case of perforated, by D'Arcy Power 14
„ „ surgical treatment of, by A. W. M. Robson 311
Gastro-enterostomy, case of, by D'Arcy Power 13
Gastro-intestinal disease, relation of epilepsy to 299
General paralysis of insane, by H. C. Thomson 119
„ „ by G. H. Savage, 321, 369
Giddiness, by F. J. Smith 306
Gonorrhœa, case of, by A. M. Sheild . . 69
Gonorrhœal epididymitis, case of, by D'Arcy Power 16
Gummatous bursitis, case of, by H. L. Barnard 188

Habershon, S. H., on bronchiectasis . 161, 183
Hæmaturia in children, by R. Hutchison . 360

PAGE

Hæmorrhoids, diagnosis and treatment of, by A. M. Sheild 273
Headache in children, by R. Hutchison . 360
Head injuries, by T. P. Legg . 137, 153, 166
Heart, enlargement of, by W. H. White . 17
„ disease, cases of, by C. W. Chapman . 349
Herschell, George, on the differential diagnosis of chronic disorders of the stomach . . 385
Hutchison, Robert, on some common symptoms of disease in children and their diagnostic significance 353
Hyoscin hydrobromate injections in morphinism 248

Immunisation with ovary 214
Impetigo, by T. C. Fox 214
Infant-feeding, modification of cow's milk for, by G. F. Still 81
Insane, general paralysis of, by Campbell Thomson . 119
„ „ by G. H. Savage, 321, 369
Itching and its treatment, by G. N. Meachen 121

Keloid on vaccination marks, case of, by H. L. Barnard 189

Lack, H. Lambert, on nasal polypus . . 8
„ on ozæna or atrophic rhinitis . . . 145
Lane, W. Arbuthnot, on the operative treatment of the conditions of the large bowel which result from chronic constipation . 209
Legg, T. P., on head injuries . 137, 153, 166
Leucocytosis, large mononuclear, in malaria, blackwater fever, and lymphadenoma, by F. Bushnell 59
Leukæmia, spleno-medullary, by F. W. Mott. 225
Liver, common diseases of, by W. H. White . 257
Locomotor ataxy, trophic changes in, by H. C. Thomson 264
Lorenz's treatment of congenital displacement of hips, results of 335
Lungs, non-tuberculous apical affections of, by H. B. Shaw 377
Lymphadenoma, large mononuclear leucocytosis in, by F. Bushnell 59

Digitized by Google

PAGE
McGavin, Lawrie, treatment of tuberculous disease of tarsus 300
Malaria, large mononuclear leucocytosis in, by F. Bushnell 59
Malignant disease of face, case of, by D'Arcy Power 16
Meachen, G. Norman, on itching as a symptom, and its treatment 121
Milk, modification of cow's, by G. F. Still . 81
Morphinism, hyoscin hydrobromate injections in 248
Morton, Charles A., diagnosis of cause of acute abdominal pain 232
Mott, F. W., on spleno-medullary leukæmia . 225

Nasal polypus, by H. L. Lack 8
Nerve injuries, by J. Sherren 53
Nerves, cranial, injury to, by T. P. Legg . 166
Neuroma of musculo-spiral nerve, case of, by H. L. Barnard 190
Nose and asthma, connection between, by A. Francis 222

Ogilvie, George, what is syphilitic disease of the nervous system? 401
Orbit, on injuries and diseases of the, by E. T. Collins. 241
Osteitis of tibia, case of, by D'Arcy Power . 16
Otorrhœa in infants, by S. Paget . . . 109
Ovarian cysts, by E. S. Bishop . . . 329
Ovary, immunisation with 214
Ozæna, by H. L. Lack 145

Paget, Stephen, on some aural cases . . 107
Pain: its importance in diagnosis, by A. Bowlby 289
Papilloma of tongue, case of, by D'Arcy Power 16
Papillomatous ovarian cysts, by E. S. Bishop. 329
Paralysis in children, by R. Hutchison . . 359
„ general, of insane, by Campbell Thomson . 119
„ „ „ by G. H. Savage 321, 369
„ peripheral facial, orbicularis sign in. 111
Pearson, S. Vere, on some rarer types of pneumonia in infants 200
Pharyngitis, chronic, treatment of . . . 53
Piles, their nature and treatment, by J. J. Clarke 177, 206

PAGE
Pneumonia in infants, by S. V. Pearson . 200
Polypus, nasal, by H. Lambert Lack . . 8
Power, D'Arcy, in Out-patient Department of St. Bartholomew's Hospital 13
„ on some cases of gastric surgery and their results . 97
Pye-Smith, P. H., on albuminuria. . . . 129
Pyrexia in children, by R. Hutchison . . 356

Rannulæ, two cases of, by H. L. Barnard . 188
Rhinitis, atrophic, by H. Lambert Lack . 145
Rib, case of supernumerary cervical, by H. L. Barnard 190
Rigidity of spine, by Sir W. Bennett . . 49
Robinson, T., is an eczema ever symptomatic? 111
Robson, A. W. Mayo, surgical treatment of gastric ulcer 311
Röntgen rays in treatment of cancer . . 267

Savage, George H., on general paralysis of the insane 321, 369
Shaw, H. Batty, non-tuberculous apical affections of the lungs 377
Sheild, A. Marmaduke, cases at St. George's Hospital . . 65
„ „ on the diagnosis and treatment of hæmorrhoids . . 273
Sherren, James, on some common nerve injuries, their examination and diagnosis . 53
Smith, Fred. J., on emphysema and bronchitis 43
„ on giddiness . . . 306
Spine, rigidity of, by Sir W. Bennett . . 49
Still, George F., on the modification of cow's milk for infant-feeding 81
Stomach, differential diagnosis of chronic disorders of, by G. Herschell . 385
„ hyperæsthesia of, by W. S. Fenwick 296
Sublimate, endo-venous injections of . . 240
„ injected into the kidney . . 352
Sugar, assimilation of, during childhood . 349
Surgery, thoroughness in, by W. W. Cheyne . 33
Swallowing, difficulty in, by H. T. Butlin . 1
Synovitis, case of gummatous, by H. L. Barnard 188
Syphilitic disease of the nervous system. What is it? by G. Ogilvie 401
Syphilitic (post-) stigma, new . . . 282

Digitized by Google

PAGE

Tabes with Charcot's joint lesion, case of, by B. Bramwell 238
Tarsus, tuberculous disease of, treatment of, by L. McGavin 300
Taylor, James, on epilepsy and its treatment . 26
Tetanus, ætiology of rheumatic 221
Thiosinamin, injections of, in Dupuytren's contracture 144
Thomson, Campbell, clinical studies in diseases of the nervous system: the trophic changes in locomotor ataxy . . 264
,, ,, general paralysis of the insane . . . 119
,, ,, injury to the cerebral cortex . . . 392
Thymus gland, by E. Cautley 395
Thyroid gland, case of parenchymatous enlargement of, by H. L. Barnard . . . 189
Tibia, osteitis of, case of, by D'Arcy Power . 16
Tinnitus, significance of, by S. Paget . . 110
Tongue, case of cancer of, by D'Arcy Power . 15
,, ,, papilloma of, by D'Arcy Power 16

PAGE

Tuberculous disease of tarsus, treatment of, by L. McGavin. 300
Tumours, broad ligament, by Alban Doran . 149

Ulcer, gastric, surgical treatment of, by A. W. M. Robson 311
,, of face, case of, by A. M. Sheild. . 70
,, perforated gastric, case of, by D'Arcy Power 14
Urine, to reduce alkalinity of 136

Veratrum viride in health and in eclampsia . 88

Wasting in children, by R. Hutchison . . 353
White, W. Hale, on common diseases of the liver 257
,, ,, some clinical considerations concerning certain of the rarer forms of enlargement of the heart . . 17
Williams, Leonard, on some points in balneary therapeutics 249

X-ray treatment of uterine cancer . . . 296

PRINTED BY ADLARD AND SON, BARTHOLOMEW CLOSE, E.C.; 20, HANOVER SQUARE, W.; AND DORKING.

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google

Digitized by Google